# THE PLANT DISEASE REPORTER

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29 MAR 1961

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Paul R. Miller

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# CONTROL OF TOBACCO BROWN SPOT BY FIELD SPRAYING WITH DYRENE

G. B. Lucas

Brown spot of tobacco, caused by Alternaria longipes, was the most important tobacco disease in North Carolina in 1959 and 1960 with losses estimated to be $21,000,000 and $9,500,000, respectively. The disease is characterized by leaf spots ranging up to 1 inch or more in diameter. As the spots enlarge the centers die and become brown. During rainy periods there may be a halo of yellow tissue surrounding the brown lesions. When the lesions are numerous they may fuse and render the entire leaf ragged and worthless. All varieties of flue-cured tobacco in use today are susceptible to brown spot. However, they differ in the amount of damage resulting from the disease.

Preliminary tests in 1958 and 1959 indicated that spraying tobacco plants with 2,4-dichloro-6-(o-chloroanilino)-s-triazine (Dyrene)[1] reduced the severity of the disease. Therefore, in 1960 spray tests were set up at three locations, one in each of three tobacco-producing areas, to determine the value of Dyrene for brown spot control.

Four commercially grown varieties, NC 73, NC 75, Coker 187 and Coker 187-Hicks, were selected and planted in 40-plant rows in randomized plots replicated at least twice. Treated plots were sprayed once, twice or three times at weekly intervals at the beginning of the harvest season (just after the plants produced flowers). Dyrene was applied with either a portable 3-gallon compressed air or a portable power sprayer at the rate of 1 pound of active material (2 pounds of 50% wettable Dyrene)/100 gallons of water using about 150 gallons/acre. All plots were separated by single rows of unsprayed unharvested plants to permit buildup of inoculum and help prevent spray drift. Tobacco from each plot was harvested and the leaves cured and graded to determine yield and quality.

By the end of the harvest season brown spot had occurred at all locations and appeared to be doing considerable damage, particularly in the unsprayed plots. Plots receiving one or two applications at weekly intervals did not consistently outyield or give higher dollar returns per acre than the unsprayed plots. However, those plots receiving three weekly applications of Dyrene had higher per-acre yields of higher quality tobacco as reflected in average price per pound (Table 1). Sprayed plots on the average yielded 230 pounds more tobacco per acre that sold 4 cents/pound higher and returned 167 dollars/acre more than the unsprayed plots. Cured tobacco of NC 73 and NC 75 was damaged less from brown spot than that of Coker 187 and Coker 187-Hicks. Apparently the first two sprays were applied too early to be effective since the disease did not become conspicuous until late in the growing season. Control might have been improved had the spray program been delayed 2 weeks. Since brown spot occurrence is dependent on the weather, being more severe in rainy seasons, proper timing and application of field sprays may be difficult.

Table 1. Increases in tobacco yields and dollar values per acre resulting from control of brown spot by spray applications of Dyrene.

| Location | Variety | Treatment | Yield (lbs/acre) av. | Yield (lbs/acre) increase | Value ($/acre) av. | Value ($/acre) increase | Price/lb ($) av. | Price/lb ($) increase |
|---|---|---|---|---|---|---|---|---|
| 1 | Coker 187 | check | 2082 | | 1200 | | .57 | |
| | | sprayed | 2220 | 138 | 1303 | 103 | .59 | .02 |
| | NC-73 | check | 1991 | | 1225 | | .62 | |
| | | sprayed | 2095 | 104 | 1334 | 109 | .64 | .02 |
| | Coker 187-Hicks | check | 2338 | | 1323 | | .57 | |
| | | sprayed | 2426 | 88 | 1401 | 78 | .58 | .01 |
| 2 | Coker 187 | check | 1571 | | 760 | | .48 | |
| | | sprayed | 1701 | 130 | 884 | 124 | .52 | .04 |
| | NC-75 | check | 1880 | | 839 | | .45 | |
| | | sprayed | 1914 | 34 | 1114 | 275 | .58 | .13 |
| 3 | Coker 187 | check | 1486 | | 510 | | .34 | |
| | | sprayed | 1974 | 488 | 668 | 158 | .34 | .00 |
| | NC-75 | check | 1072 | | 392 | | .37 | |
| | | sprayed | 1704 | 632 | 712 | 320 | .42 | .05 |
| Average | | | | 230 | | 167 | | .04 |

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[1]Kindly furnished by Chemagro Corporation, Box 4913, Hawthorn Road, Kansas City, Missouri.

# RHIZOCTONIA FRUIT ROT OF PROCESSING TOMATOES[1]

W. T. Schroeder and R. Provvidenti

## Summary

An unusual development of Rhizoctonia fruit rot occurred among processing tomatoes in western New York during an abnormally dry harvest season in 1960. Water-soaked lesions, ranging from small circular spots to large irregular portions of the fruit, were more characteristic on the ripe processing fruit than were the brownish, sunken and zonate lesions described for the disease as it occurs on shipping or market tomatoes in the mature green or light pink stage. Inoculation studies showed that the type of symptoms was a function of fruit maturity at the time of infection. Evidence obtained indicated the organism penetrated only indirectly through epidermal injuries. Mold count studies indicated that Rhizoctonia fruit rot might be just as important a contributor as anthracnose.

## INTRODUCTION

Rhizoctonia fruit rot of tomato, also known as soil rot, has been long recognized as a serious transit disease of market or green-wrap tomatoes (2, 3, 4, 5), but its importance on processing tomatoes has not been stressed. An unusually abundant development of the disease was observed in fields of processing tomatoes in western New York during the 1960 season. The symptoms and conditions under which they developed were somewhat different from those described for market tomatoes by the earlier investigators and suggest that the disease may not always have been recognized on fruits for processing.

In surveys made in commercial fields from September 20 through October 20, 1960, the percentage of infected red ripe fruit ranged from a trace in some fields to as high as 26% in others. In many fields Rhizoctonia rot exceeded anthracnose in importance. The greatest incidence occurred in planting on light sandy soils. Although Doolittle (1) and others have indicated that infections are most common during periods of heavy rains and on resulting wet soils, the outbreak in western New York occurred during one of the driest harvest seasons on record. Furthermore, infected fruits were as common high on the plant away from the soil as they were on or near the soil. Symptoms were not typical of those described and emphasized for the disease on the green-wrap crop.

## SYMPTOMS ON RIPE FRUIT

Symptoms on naturally infected ripe fruit were somewhat variable, the most characteristic being a water-soaked condition of the fruit ranging from small circular spots to large irregular areas involving half or more of the fruit surface (Fig. 1A, B, and C). Water-soaked areas were always dark red in color.

Zonations or mottled areas suggestive of buckeye rot (Phytophthora sp.) or Rhizoctonia rot as described for green-wrap tomatoes were not observed on pink or red fruit in the field, except in two instances. In neither of these instances was Rhizoctonia isolated from the lesions.

Some fruits were found occasionally with a small dark zone in the center of a circular lesion which was presumably the point of entry (Fig. 1C), but in most fruits not detectable points of entry were evident (Fig. 1B). The water-soaked lesions were initially suspected to be bacterial infections, but after the epidermis was peeled away an uncharacteristic firmness of the tissue was evident. Rhizoctonia was the sole organism in the majority of the isolations from such lesions. Of platings made from 75 fruits, 65 yielded pure cultures of Rhizoctonia sp., 5 Alternaria sp., 2 Colletotrichum sp., 1 Fusarium sp. (zonate lesions) and 2 yielded unidentified fungi (one of which came from the other zonate lesion previously mentioned).

## INOCULATION STUDIES

The results of artificial inoculations of detached green, pink, and red tomato fruits confirmed some of the previously published observations on Rhizoctonia fruit rot, but raised cer-

[1]Approved by Director of the New York State Agricultural Experiment Station, Geneva, New York as Journal Paper No. 1246.

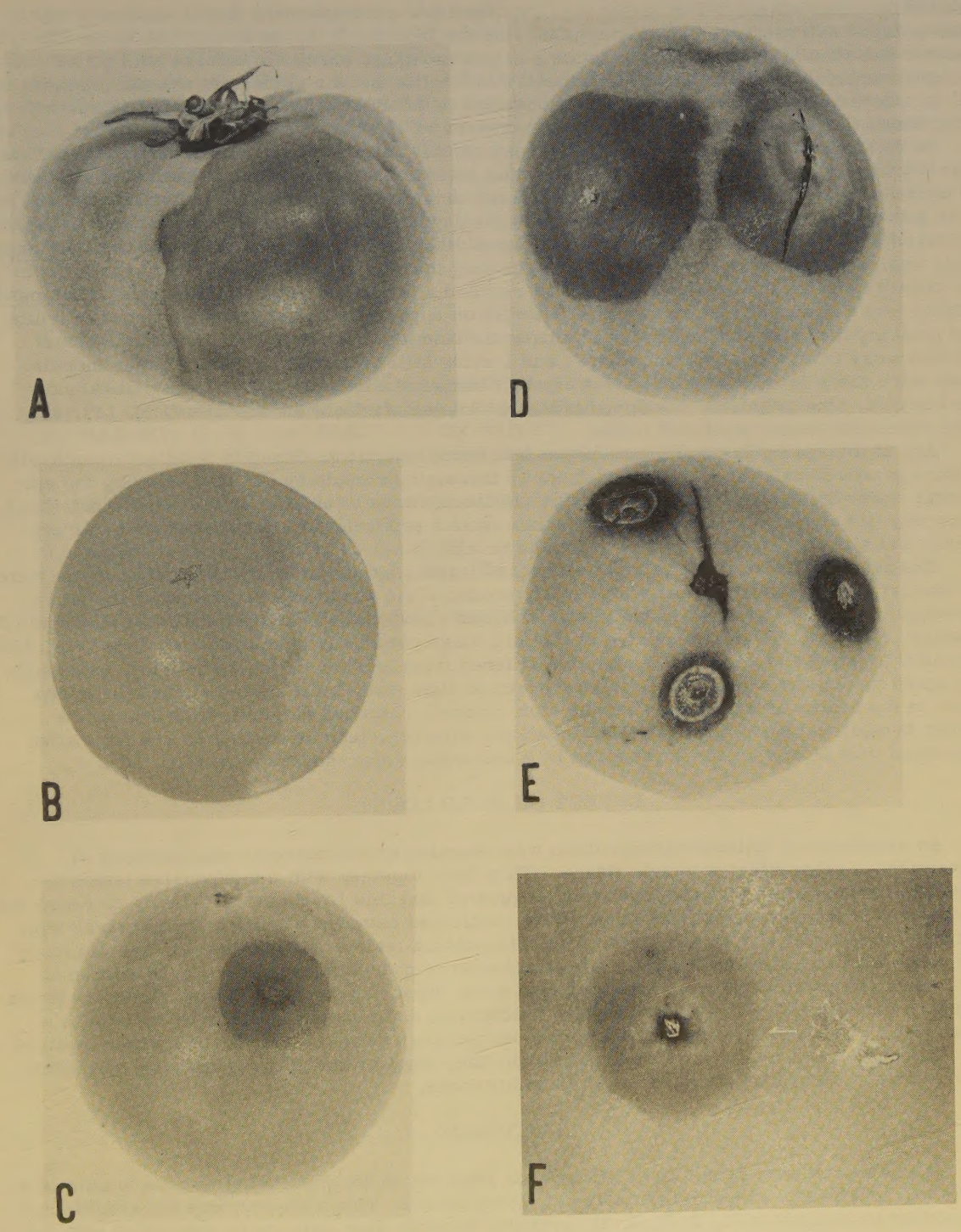


FIGURE 1. Tomato fruits infected by Rhizoctonia sp. A and B -- Naturally infected red fruit showing extensive lesions with no visible point of entry. C -- Naturally infected fruit showing circular lesion with point of entry in the center of dark area. D -- Fruit artificially inoculated at pink stage and showing marked zonations on lesion to the right and lack of zonations on lesion to the left. E -- Green fruit artificially inoculated showing typical brownish zonations. F -- Fruit infection through remote prick in epidermis; mycelial biscuit (arrow) above lesion and 1/2 inch away.

tain doubts about others. Two methods of inoculation were employed. In one method a mycelium-potato-dextrose agar biscuit was placed on the fruit surface; the epidermis immediately beneath was then injured by lightly pricking it just enough to break the surface with a fine-pointed sterilized needle. The other method was similar but the epidermis was not pricked. All inoculated fruits were held in a moist chamber at 80° F and 100% humidity for 48 hours, after which they were removed to room temperature of 75° to 80°.

In the initial experiments infections developed only when the epidermis was injured. This result was confirmed in another experiment that involved 16 inoculations of injured fruits and 16 of non-injured fruits. Within 48 hours infections were observed on the injured fruits, but none was observed on the non-injured fruits. Six days later, small lesions of various shapes appeared on the non-injured fruits at points some distance from the mycelial biscuits. Rhizoctonia was recovered from these lesions, suggesting that the organism might have grown from the biscuit over the intact epidermis until it reached a break and entered through it. This possibility was checked by placing mycelial biscuits on apparently sound greenhouse-grown fruits and pricking the epidermis immediately under the biscuits on some, pricking the epidermis 1/2 inch away from the biscuit on others, and leaving still others uninjured. Fifteen inoculations were made in each of the above groups. Fourteen lesions developed from pricks under the biscuits, nine infections developed in the pricks remote from the biscuits (Fig. 1F), and none developed on the uninjured fruits.

Age of the agar culture of the pathogen influenced infection. Biscuits from a 2-month-old culture produced only three infections from 15 inoculations made by directly pricking the epidermis under the biscuit, and none from the 15 inoculations attempted by the remote prickings. Four-day-old mycelial biscuits from the same isolate produced 14 infections from 15 direct pricks and nine infections from the 15 remote pricks.

The nature of the symptoms was determined largely by the maturity of the fruit at the time of inoculation. Immature green fruit usually produced the symptoms described by the early investigators (1, 5), namely, slightly sunken brown spots which become zonate in some instances (Fig. 1E). Fruits inoculated at the pink stage commonly developed zonations (Fig. 1D). Frequently symptoms from one inoculation differed from another on the same fruit, especially in regard to zonations (Fig. 1D). Mature green or light-pink fruits often retained the green color in the center of the lesion when the fruit ripened. Lesions on green fruit required a longer incubation period and developed zonations; whereas, lesions on pink or red fruit often developed only a dark center surrounded by a uniformly water-soaked area (Fig. 1C).

## EFFECT ON MOLD COUNT

An abundance of Rhizoctonia mycelium was revealed by microscopic examinations of smears made from infected tomato tissue. This fact, together with the expansive lesion development observed on infected ripe fruit, suggested that this rot might materially increase the mold count of the processed fruit juice. Five replicated samples of juice were prepared with weighed increments of diseased tissue from Rhizoctonia lesions and another set was similarly prepared from anthracnose lesions. All samples were passed through a standard screen to simulate a commercial juice process and then were submitted for mold counts according to the standard procedure[2]. Mold counts for the Rhizoctonia samples ranged from 40 to 76 with a mean of 61, whereas those from the anthracnose samples ranged from 23 to 60 with a mean of 42. Although these counts are abnormally high, they suggest that Rhizoctonia may contribute as materially toward a high mold count as anthracnose.

## DISCUSSION

The characteristic symptom of Rhizoctonia fruit rot of the processing or ripe tomato is a dark red, water-soaked and irregularly advancing lesion. This symptom was not emphasized by earlier investigators (1, 2, 3, 4, 5), probably because they were more concerned with the disease on market tomatoes which are picked at a different stage of maturity -- the mature green or light-pink stage. Fruits infected at that stage of development usually show the zonate lesions which may develop into a general rot when the fruit ripens in transit. Red fruits, however, seldom if ever show any zonations when infected at the late-pink or red-ripe stage of maturity.

[2]The assistance of Mrs. Margaret N. Albury, Department of Food Science and Technology, in making the mold counts is gratefully acknowledged.

Heavy rains and wet soil, suggested as conducive to heavy infection by Ramsey and Bailey (3), did not appear essential for the development of Rhizoctonia fruit rot on processing tomatoes since the outbreak in New York during 1960 occurred during one of the driest harvest seasons on record. The fact that half or more of the field-infected fruits were not near the soil suggests infection by spores. The evidence obtained in these studies of penetration through wounds agrees with the earlier work of Rosenbaum (4), but disagrees with the direct penetration views of Ramsey and Bailey (3) and Weber and Ramsey (5). The apparent necessity of epidermal injuries for penetration may explain why the disease appeared in higher amounts on light sandy soils. The water-soaked condition of many of the fruits, at first mistaken for bacterial infections, suggests that Rhizoctonia might be more important on the processing crop than was previously considered, especially in view of the effect on the mold count.

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# AN ATTEMPT TO CONTROL ROOT-KNOT NEMATODE WITH DACTYLARIA THAUMASIA AND ARTHROBOTRYS ARTHROBOTRYOIDES

R. Mankau

## Abstract

Attempts to control or significantly reduce root-knot damage to tomato and okra by inoculating the predacious hyphomycetes Dactylaria thaumasia Drechsler and Arthrobotrys arthrobotryoides Lindau into soil infested with Meloidogyne incognita were unsuccessful. Soil amendments of wheat germ, straw and steer manure gave better plant growth but did not appear to aid the activity of the fungi.

## INTRODUCTION

Several attempts have been made to utilize nematode-trapping fungi to control populations of plant-parasitic nematodes in the soil. Linford and Yap (4) reported that root-knot nematode injury was significantly reduced by Dactylella ellipsospora while four other fungi (Arthrobotrys oligospora, A. musiformis, Dactylaria candida and D. thaumasia) failed to reduce nematode damage. Hutchinson and Mai (3) found that Dactylaria eudemata did not exert any control on Heterodera rostochiensis in limited soil tests. The addition of three forms of organic matter, with and without Dactylaria thaumasia by Duddington, et al. (2) produced no significant effects upon the cyst and egg populations of the golden nematode of potato or upon potato yields. The same authors (1) tested the effect of Dactylaria thaumasia and organic matter upon a population of Heterodera schachtii and the yield of sugar beets. The final nematode populations were not significantly affected, and although some of the treatments increased beet yield somewhat, this effect could not be attributed solely to the fungus application.

This report describes similar attempts to test the use of predacious fungi against a nematode pest.

## EXPERIMENTAL

Two fungi were used in all of the tests, Arthrobotrys arthrobotryoides Lindau and Dactylaria thaumasia Drechsler. The former was isolated from a citrus grove soil near Olive, Orange County, California, while the latter was isolated from a soil sample obtained from Florida. Both fungi grow well on a variety of laboratory media, but conidial dimensions and growth most typical of actively trapping mycelia in soil are obtained on weak corn meal agar. Large quantities of the fungi for inoculation into soil were produced in liquid culture of a 2% wheat germ extract. These fungi do not produce trapping organs in pure culture. In the presence of nematodes, however, traps are formed abundantly.

On agar plates to which either of these fungi and a mixture of nematodes are added a large number of the nematodes are captured. The efficiency of the trapping is never 100%, however. Adult Rhabditis sp. and nemas of about 1 mm or larger often pull loose from the traps and escape. Citrus nematode larvae, root-knot larvae and nemas such as Aphelenchus sp. and Aphelenchoides sp. are more easily captured.

Test I: A preliminary test was conducted in which the soil was prepared by mixing a sterile greenhouse potting mix with soil from greenhouse stock cultures of the root-knot nematode, Meloidogyne incognita, in approximately a 1:1ratio. The soil was dispensed to eighteen 6-inch clay pots and three treatments were set up. Treatment A was inoculated with Arthrobotrys arthrobotryoides and Treatment B with Dactylaria thaumasia. Treatment C was an uninoculated check. Mycelial mats from broth cultures of the fungi were collected on filter paper in a Büchner funnel, combined with distilled water and blended in a blender for 30 seconds. Twenty-five ml of the resulting suspension was then added to each pot to be inoculated with a syringe. Young tomato seedlings about 2 inches high were planted in each pot. Superimposed upon treatments A and B was the addition of 10 g of wheat germ as an amendment in three of the six pots.

The plants were grown for 83 days on a greenhouse bench and then harvested. Fresh top weights and dried root weights are recorded in Table 1. Pots receiving the predacious fungi had decidedly better growth of the tomato plants than did the checks. Somewhat better growth occurred where the wheat germ amendment was added.

From the results of this test, apparently the addition of the two species of nematode-trapping fungi to the soil resulted in a distinct reduction in the severity of root-knot attack. At the end of the test, soil from each pot was examined for the presence of nematode-trapping fungi by

Table 1. Fresh top weights and dried root weights (grams) of tomatoes in soils inoculated with predacious fungi (Test I).

| | | A Arthrobotrys arthrobotryoides | | B Dactylaria thaumasia | | C[a] Control | | |
|---|---|---|---|---|---|---|---|---|
| | Rep. | top wt. | root wt. | top wt. | root wt. | | top wt. | root wt. |
| I Wheat germ amendment | 1 | 188 | 4.7 | 152 | 2.8 | 1 | 34 | 0.7 |
| | 2 | 185 | 2.7 | 192 | 2.7 | 2 | -[c] | -[c] |
| | 3 | 228 | 3.3 | 141 | 2.7 | 3 | -[c] | -[c] |
| II No amendment | 1 | 107 | 2.9 | 75 | 1.1 | 4 | -[c] | -[c] |
| | 2 | 145 | 3.7 | 169 | 3.8 | 5 | 95 | 3.7 |
| | 3 | -[b] | -[b] | 74 | 1.3 | 6 | 21 | 0.4 |

[a] None of the controls received the wheat germ amendment.
[b] Plant died due to root rot, complicated by nema infestation.
[c] Plants died, due to heavy root-knot infestation.

plating the soil on 1/4 strength corn meal agar. A. arthrobotryoides was not recovered from one pot inoculated with this fungus, while D. thaumasia was not recovered from 50% of the pots to which this fungus was introduced. Most of the pots, however, contained a large number of other species of nematode-trapping fungi, some of which are more effective in trapping nematodes than the two inoculated species. These species included: Arthrobotrys dactyloides Drechsler, Arthrobotrys sp., Dactylella ellipsospora Grove, D. gephyropaga Drechsler, and Dactylaria brochopaga Drechsler. Species other than those inoculated were recovered from every pot in the test except one of the remaining check plants from which no nematode-trapping fungi were recovered.

Test II: Both of the fungus species used in Test I were also used in an outdoor microplot test. A Meloidogyne incognita population had been built up in the soil of the test area by previous croppings. The area consisted of a 15 x 30 foot cubicle circumscribed by steel plate sunk approximately 3 feet into the soil, and was separated into 7 1/2 x 6 foot microplots by corrugated metal sheets sunk about 18 inches into the soil. Four treatments and a check were set up as indicated in Table 2. The straw mulch and steer manure were applied at a rate of 20 pounds and 39 pounds per microplot, respectively, and were worked into the soil thoroughly by turning the soil several times with a shovel and raking. A winter cover crop of vetch was planted in the microplots after inoculation of plots receiving fungi with mycelial suspensions prepared by blending cultures of the aforementioned species and injecting into the soil with a large steel syringe in a 12 x 15 inch grid pattern. In the spring the vetch was removed and the same fungi were again inoculated into the plots of treatments B and E. On May 18 okra seed was planted in hills spaced 18 inches apart in the microplots. Harvest records on okra pod production were begun for each plot in July and followed for 2 months. Results are given in Table 2, along with average counts of root-knot larvae extracted from the soil in each treatment.

Table 2. Outdoor microplot test. Averages of okra pod harvest (July 22 - Sept. 25) and root-knot larvae counts per treatment (Test II).

| Treatments | Okra pods (grams) | Larvae/50 cc soil |
|---|---|---|
| A. Straw mulch | 4950 | 236 |
| B. Straw mulch + fungi | 4499 | 231 |
| C. Check (untreated) | 4325 | 427 |
| D. Steer manure | 7627 | 153 |
| E. Steer manure + fungi | 6170 | 925 |

No significant differences in okra pod production occurred in the plots receiving nematode-trapping fungi. The treatments receiving manure had significantly better growth due solely to the addition of the manure. A count made of root-knot larvae present in the soil in each plot revealed nothing which could be related to the action of the introduced fungi.

Individual plants in treatments D and E which made good growth had severely galled roots supporting large numbers of mature females. Some plants in all treatments were eventually killed by nematode attack. No control of the nematodes was demonstrated in this test.

## DISCUSSION

The predacious fungus species used in the tests are both types which trap nematodes in three-dimensional adhesive hyphal networks and are similar to those used by other authors (1, 2, 3, 4). It is unlikely that this type of predacious fungi can afford an adequate level of protection in dicotyledonous plants such as okra and tomato where the larval progeny of a single nematode may multiply within heavily galled roots in which the fungi cannot reach them. The introduction of the relatively large quantity of predacious fungus initially into treatments A and B of the greenhouse test may have been a factor in reducing the larval population to an extent which gave better growth of the plants; nevertheless, some plants in these treatments were heavily galled and all had some degree of root-knot infestation. The addition of wheat germ as a soil amendment probably served as a fertilizer for the tomato plants as well as or rather than a stimulation to fungus activity. No check was provided for this factor.

The somewhat beneficial results obtained in the greenhouse test could not be attributed solely to the fungi introduced into the treatments, since many other species of predacious fungi were also present in the soil. Some predacious fungi probably occur in all non-sterile soils and must be taken into account in evaluating given species under field conditions.

The result of the outdoor test has discouraged any further trials with these particular species.

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# RESULTS OF FUNGICIDE TESTS FOR THE CONTROL OF WALNUT ANTHRACNOSE, 1960

Frederick H. Berry[1]

Walnut anthracnose, caused by the fungus Gnomonia leptostyla (Fr.) Ces. & de N., is the most serious pest of eastern black walnut (Juglans nigra). The disease first appears in late May or early June and is found at any time thereafter until the leaves have been shed. Anthracnose is much more prevalent during rainy seasons. Under environmental conditions favorable to development of the fungus, many black walnut trees are severely defoliated by mid-summer. This premature defoliation often results in poorly filled, low-quality nuts. The disease also reduces growth, and greatly weakens the trees.

Previous experiments have shown that four applications of a protective fungicide, such as zinc ethylene bisdithiocarbamate (zineb) (Dithane Z-78) or manganese ethylene bisdithiocarbamate (maneb) (Dithane M-22), are necessary to control anthracnose[2]. To protect the trees from primary infection, it is necessary to apply the first spray before ascospore discharge takes place. The foliage on most black walnut trees is one-half to three-fourths mature size at this time. Then three additional sprays should be applied at approximately bi-weekly intervals. Zineb and maneb are applied at the rate of 2 pounds of fungicide in 100 gallons of water.

During the 1960 field season tests were made to determine if the addition of a spray adhesive increased the efficacy of fungicides in controlling walnut anthracnose. Twenty-seven eastern black walnut trees of six different varieties were used in the experiment. These trees, approximately 30 years old, were all growing in the same orchard at the Plant Industry Station, Beltsville, Maryland.

The trees were sprayed with zineb or maneb alone and with Good-rite p.e.p.s., a spray adhesive, added to the fungicides. The fungicides were applied at the rate of 2 pounds/100 gallons of water and the spray adhesive at the rate of 1/2 pint/100 gallons of water. Spray applications included S-(1,2-bis(ethoxycarbonyl)ethyl)O,O-dimethyl phosphorodithioate (malathion) at the rate of 1 1/2 pounds/100 gallons of water for insect control. Certain trees were left as controls and sprayed with water and insecticide only. Sprays were applied on June 6 and 17, and July 6 and 20 with a hydraulic sprayer that developed approximately 600 pounds of pressure at the spray gun.

Weather conditions favorable for anthracnose development prevailed throughout the 1960 growing season, and consequently infection was severe. A rain gauge maintained at the orchard indicated the following precipitation: June, 1.62 inches; July, 5.01 inches; August, 5.29 inches; and September, 5.13 inches. The data on control of the disease reported in Table 1 was recorded on September 19, 1960.

The results in Table 1 indicate that the addition of a spray adhesive, such as Good-rite p.e.p.s., to maneb and zineb did not increase the effectiveness of these fungicides in controlling walnut anthracnose. Although the data were taken quite late in the season, less than one-fourth of the leaflets on the trees that received any of the four spray treatments were infected. The unsprayed control trees were severely defoliated and the leaflets remaining on these trees were all infected with anthracnose.

Table 1. Relative effectiveness of fungicides with and without a spray adhesive in control of walnut anthracnose, Beltsville, Maryland, 1960.

| Materials | Number trees treated | % infection of leaflets[a] | Defoliation rating[b] |
|---|---|---|---|
| Zineb | 6 | 22 | 2.7 |
| Maneb | 6 | 17 | 2.3 |
| Zineb plus Good-rite p.e.p.s. | 6 | 19 | 2.5 |
| Maneb plus Good-rite p.e.p.s. | 6 | 17 | 2.2 |
| None (control)[c] | 3 | 100 | 5.0 |

[a]Average infection based on 100 leaflets per tree.

[b]Defoliation rating as follows: None, 0; very light, 1; light, 2; considerable, 3; heavy, 4; very heavy, 5; complete, 6. Average defoliation rating for the trees receiving the various treatments is given.

[c]Sprayed with water and insecticide.

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# DYRENE PHYTOTOXICITY EFFECTS ON TOMATO[1]

John Paul Jones[2]

## Abstract

During the fall crop season of 1960 all spray mixtures containing 2,4-dichloro-6-(o-chloroanilino)-s-triazine (Dyrene) caused injury to Homestead 24 tomato plants. The combinations of Dyrene (2 pounds/100 gallons) + Tribasic Copper Sulfate (4 pounds) + parathion + DDT + Tecmangam and Dyrene (2 pounds) + parathion + DDT caused extreme leaflet crinkling and distortion, leaf and stem necrosis, slight plant stunting, shallow pitting of the fruit pericarp, and decreased fruit numbers. No decrease in fungicidal value, as determined by gray leafspot (Stemphylium solani Weber) control estimates, was apparent.

## INTRODUCTION

Excellent control of several tomato foliage diseases has repeatedly been demonstrated with 2,4-dichloro-6-(o-chloroanilino)-s-triazine (Dyrene). In combination with manganese ethylene bisdithiocarbamate (maneb) Dyrene gives effective control of all of the major fungus foliage diseases (1, 2, 5, 7). For this reason, the use of the compound alone or in combination with other fungicides has greatly increased in Florida.

Although no Dyrene injury was observed by the author in previous spray trials (3), nor has any serious injury been reported elsewhere, there have been indications that Dyrene alone or in combination with other fungicides was phytotoxic to tomato plants. Stall (6) demonstrated that Dyrene in certain ratios with Tribasic Copper Sulfate (TBCS) caused a slight leaf crinkle on tomato plants. According to Stall this injury only existed during a high temperature period and thereafter was not noticeable. On the west coast of Florida Walter (8) observed that both greenhouse and field tomatoes sprayed with Dyrene or Dyrene combinations occasionally exhibited injury manifested as leaf necrosis and distortion, and flower burn.

Because of the increased use of Dyrene by west coast growers and the possibility of Dyrene phytotoxicity a field trial was carried out during the fall of 1960 to determine if Dyrene and certain Dyrene combinations were phytotoxic to tomatoes.

## MATERIALS AND METHODS

All fungicide combinations used, with but one exception, were those recommended for Florida growers and are currently in commercial use (Table 1). The additional combination was that of Dyrene + TBCS which is used by some growers during the early fall for bacterial spot (Xanthomonas vesicatoria (Doidge) Dows.) control.

Table 1. Rates (in pounds/100 gallons) of commercial materials used in the various spray combinations.

| Treatment[a] | Dyrene 50W | Maneb 80W | Zineb 65W | TBCS 53S | Parathion 15W | Diazinon 25W | DDT 50W | Tecmangam 75W $MnSO_4$ |
|---|---|---|---|---|---|---|---|---|
| 1A | - | 1.5 | - | - | 2.0 | - | 2.0 | - |
| B | - | - | 2.0 | - | 2.0 | - | 2.0 | - |
| 2 | 1.0 | 1.0 | - | - | 2.0 | - | 2.0 | - |
| 3 | 2.0 | - | - | - | 2.0 | - | 2.0 | - |
| 4 | 2.0 | - | - | - | 2.0 | - | 2.0 | 2.0 |
| 5 | 1.0 | - | - | 4.0 | 2.0 | - | 2.0 | 2.0 |
| 6 | 1.0 | - | - | 4.0 | 2.0 | - | 2.0 | - |
| 7 | 2.0 | - | - | - | - | 1.0 | 2.0 | - |
| 8 | 1.0 | 1.0 | - | - | - | 1.0 | 2.0 | - |

[a]Treatment 1 consisted of maneb alternating with zinc ethylene bisdithiocarbamate (zineb).

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Since west coast Florida growers include fungicides, insecticides, and minor elements in their spray programs and usually mix all in the same tank, parathion[3], Diazinon[3], DDT[3], and Tecmangam were used in combination with the fungicides. Nu-iron and zinc sulfate were applied separately to all plants as needed. One week prior to fruit harvest all plants were given an extra parathion spray each week in order to control an outbreak of serpentine leaf miners (Agromyza pusilea Meigen). After the fourth harvest the extra parathion sprays were applied to all plants twice weekly.

A randomized block design with five replications was used. Spraying was initiated September 6, one week after the plants (variety Homestead 24) were set in the field, and was continued twice weekly throughout the season. All combinations were applied at the same rates (98 gallons/acre increasing to 196 gallons/acre) with knapsack sprayers.

Ratings of foliage injury were made October 13 and November 18. Gray leafspot (Stemphylium solani Weber) ratings were made December 2 by counting the number of lesions from 10 randomly selected leaflets (1 leaflet per plant) from each plot.

Flowers of one inflorescence per plant of 10 randomly selected plants per plot were counted November 15. Each inflorescence was then tagged. The number of fruit from these tagged inflorescences was counted December 9, thereby giving an estimate of the fruit setting percentage.

Harvesting of pink and ripe fruit was commenced November 22. The final harvest was made December 16 when the plants were stripped of all fruit, regardless of size or maturity, since injury was still occurring on the very young fruit.

## RESULTS

Foliage injury was noticed October 6 and was characterized by leaflet distortion (puckering and crinkling was so severe on many plants as to cause splitting of the blade), green color intensification in many leaflets, occasional leaf and stem necrosis, slight plant stunting, and flower drop (Fig. 1). These symptoms not only persisted but increased in severity throughout October and most of November. Symptoms did not develop on new foliage after the third week of November.

Foliage injury was not noticed on the maneb-zineb (treatment 1) control plants (Table 2). The most severe foliage injury occurred on plants receiving Dyrene + TBCS (treatments 5 and 6) and the least, with the exception of the controls, on plants receiving treatments 2, 7, and 8.

Table 2. The effects of certain spray treatments on fruit set and on the occurrence of foliage spray injury, gray leafspot and serpentine leaf miner control.

| Treatment | Spray injury | | Gray leafspot | Serpentine leaf miner | % fruit set |
|---|---|---|---|---|---|
| | Oct. 13 | Nov. 18 | | | |
| 1 | 1.0[a] | 1.0 | 1518[b] | 315[c] | 45.0[d] |
| 2 | 2.2 | 2.6 | 210 | 276 | 39.6 |
| 3 | 1.4 | 3.4 | 286 | 308 | 30.2 |
| 4 | 1.6 | 3.6 | 252 | 286 | 37.6 |
| 5 | 3.2 | 4.2 | 507 | 229 | 29.2 |
| 6 | 2.2 | 4.2 | 261 | 229 | 36.7 |
| 7 | 1.6 | 2.0 | 377 | 261 | 28.5 |
| 8 | 1.4 | 2.0 | 175 | 226 | 29.5 |
| LSD 05 total | | | 93.97 | 12.98 | |
| D 05 means | | | 53.13 | 7.29 | |

[a]Based on a rating of 1-5 where 1 = no injury and 5 = severe injury.
[b]Total number of lesions from 50 leaflets (10 leaflets from each of 5 replicates).
[c]Total of 40 (8 plants from each of 5 replicates) defoliation estimates based on the Horsfall-Barratt system.
[d]Percentages were converted to arc sin angles for analysis.

Fruit injury, consisting of a scattered shallow pitting of the pericarp similar to sand blast damage, was first noticed October 18 and was most apparent on exposed fruit (Fig. 2). This

[3]Parathion=O, O-diethyl O-p-nitrophenyl phosphorothioate; Diazinon=O, O-diethyl O-(2-isopropyl-4-methyl-6-pyrimidyl)phosphorothioate; DDT=1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane.

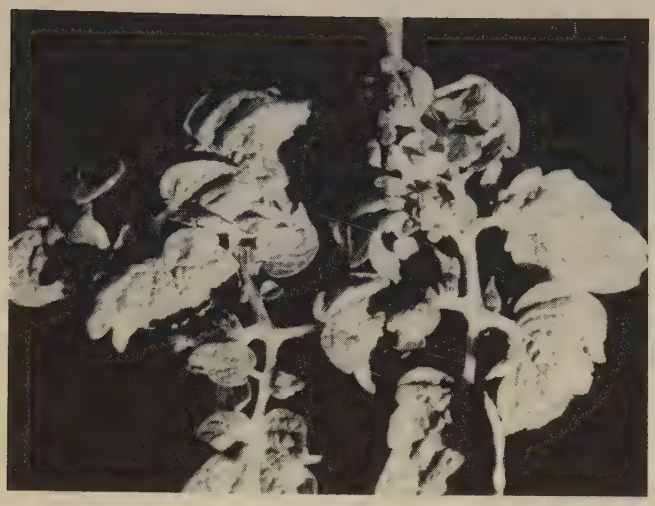

FIGURE 1. Dyrene phytotoxicity symptoms on foliage.

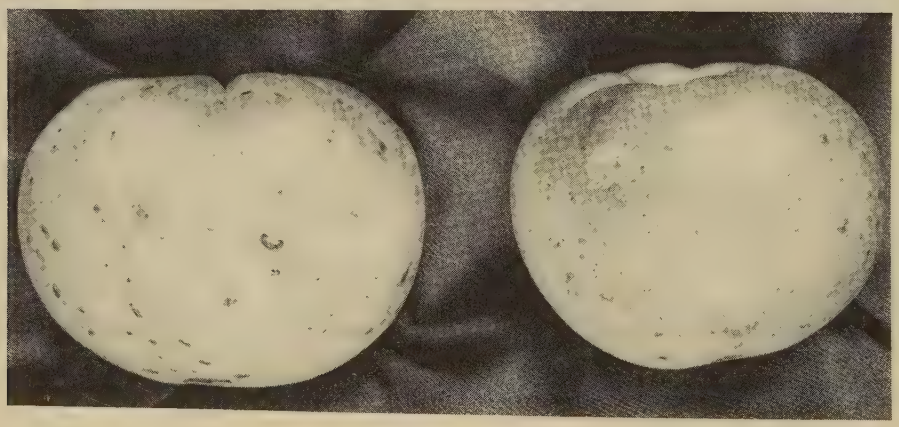

FIGURE 2. Dyrene injury on mature fruit.

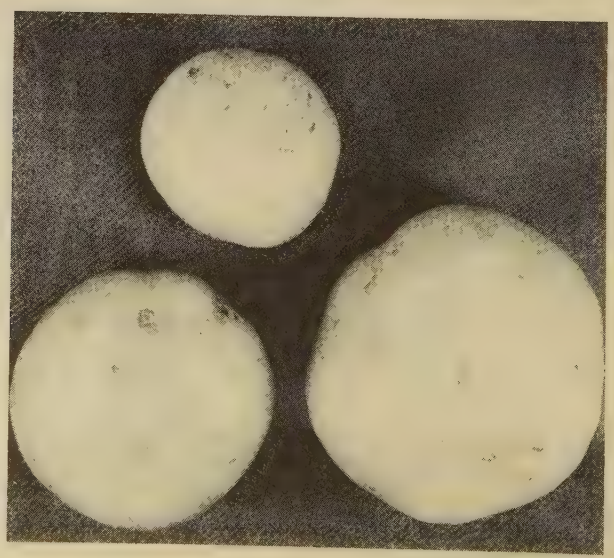

FIGURE 3. Dyrene injury on immature fruit of last harvest. Fruit sizes are: 3 x 2.5 cm, 5 x 4.5 cm, and 5.5 x 3.0 cm.

fruit damage was of culling severity and persisted throughout the season. Many of the very small immature fruit of the last harvest were severely injured (Fig. 3).

Fruit from control plants exhibited no definite Dyrene spray injury and on only approximately 1.0% was there any indication of spray injury (Table 3). Treatment 3 caused the highest incidence of fruit injury whereas treatment 4 caused the least. Despite the occurrence of definite Dyrene spray injury on a few fruit from plants receiving treatment 4, no statistical difference, according to the Tukey confidence interval, existed between this treatment and the control. Both treatments 5 and 6 caused severe fruit injury. However, treatment 5 was slightly less injurious than treatment 6. This was especially noticeable during the early harvests (Table 3). Fruit from plants receiving treatments 2 and 7 were only slightly injured and those from plants receiving treatment 8 were moderately injured.

The incidence of injured fruit decreased throughout the harvest period on all plants except those receiving treatment 3 (Table 3).

Table 3. Total fruit yields and percentages of Dyrene-injured fruit.

| Treatment | Total number fruit | % fruit injured[a] | Number fruit of 1st 4 harvests | % fruit injured of 1st 4 harvests[a] |
|---|---|---|---|---|
| 1 | 2878 | 1.1 | 942 | 1.7 |
| 2 | 3055 | 3.8 | 834 | 6.8 |
| 3 | 2622 | 19.1 | 955 | 15.2 |
| 4 | 2824 | 2.3 | 1005 | 2.8 |
| 5 | 2594 | 13.9 | 709 | 21.1 |
| 6 | 2997 | 10.1 | 859 | 14.4 |
| 7 | 3098 | 7.7 | 1062 | 11.0 |
| 8 | 3054 | 3.1 | 796 | 4.3 |
| LSD 05 | 144.83 | 0.25 | 90.72 | 0.24 |
| D 05 | 81.31 | 1.91 | 54.97 | 1.90 |

[a]Percentages were converted to arc sin angles for statistical analysis.

Because of the occurrence of severe serpentine leaf miner damage (Table 2) in certain plots and the subsequent inability of the infested plants to size fruit, only fruit numbers were compared. Lowest yields were from plants receiving treatments 3 and 5 (Table 3). All other yields were approximately equivalent.

An analysis of variance of the fruit setting percentages did not yield a significant difference among treatments (Table 2). However, a comparison of the maneb-zineb treatment versus all other treatments yielded a significant difference (4). Hence the maneb-zineb plants set a greater percentage of fruit from tagged inflorescences than the Dyrene plants taken collectively.

Poorest gray leafspot control resulted from the maneb-zineb treatment. Next poorest was the mixture of Dyrene + TBCS + Tecmangam which caused severe foliage and fruit injury. All other treatments were approximately equivalent and better than treatments 1 and 5.

Temperatures throughout most of the fall crop season were high and precipitation was low (Table 4).

Table 4. Temperature and precipitation during the fall crop season.

| Month | Temperature (°F) range | av. high | av. low | Precipitation (in inches) |
|---|---|---|---|---|
| September | 68-94 | 89.0 | 71.8 | 15.97 |
| October | 57-92 | 86.8 | 67.2 | 2.78 |
| November | 52-87 | 82.1 | 59.5 | 0.00 |
| December 1-16 | 34-83 | 72.2 | 49.0 | 1.47 |

## DISCUSSION

All spray mixtures containing Dyrene caused some foliage and fruit injury. Those mixtures combining Dyrene + TBCS were extremely phytotoxic, as were those containing 2 pounds of Dyrene without Tecmangam. Tecmangam when added to 2 pounds of Dyrene greatly decreased the incidence of fruit injury whereas Tecmangam added to the mixture of Dyrene + TBCS slightly increased fruit injury incidence.

Comparisons of total fruit numbers indicated that the mixtures (2 pounds Dyrene + parathion + DDTand 1 pound Dyrene + 4 pounds TBCS + parathion + DDT + Tecmangam) which caused the most fruit damage also decreased fruit set.

Since all of the Dyrene treatments gave much better control of gray leafspot than the maneb-zineb treatment, the fungicidal activity of Dyrene was not apparently decreased to any extent by any of the test combinations.

The fact that Dyrene can cause severe foliage and fruit injury has been established. However, the conditions under which this injury can occur remain to be determined. In general the incidence of fruit injury decreased throughout the harvest period (Nov. 22 - Dec. 16). Temperatures throughout September and October were high and only gradually decreased through November. On December 1 temperatures sharply declined and remained low throughout the remainder of the harvest period. Perhaps a correlation exists between Dyrene fruit injury and temperature, but the fact remains that the incidence of fruit injury increased throughout the harvest period on plants sprayed with 2 pounds Dyrene + parathion + DDT. Moreover, the very immature fruit of the last harvest from plants sprayed with 2 pounds Dyrene + parathion + DDT were severely injured and must have been exposed only to mild-low temperatures. Upon examination of the precipitation data, the hypothesis might be made that the injury resulted from an accumulation of spray materials. Only 2.78 inches of rain were recorded for October and none for November. But again the severe injury occurring on the very immature fruit, upon which only a few sprays could have been applied, tends to negate the residue accumulation hypothesis.

Since Dyrene gives such excellent control of several tomato foliage diseases and is currently being widely used, further phytotoxicity tests are certainly warranted to determine the conditions under which Dyrene can cause injury.

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# INFLUENCE OF STING NEMATODE CONTROL WITH O,O-DIETHYL O-2-PYRAZINYL PHOSPHOROTHIOATE ON YIELD AND QUALITY OF PEANUTS

J. N. Sasser and W. E. Cooper

## Summary

Surface applications of a 10% granular formulation of O,O-diethyl O-2-pyrazinyl phosphorothioate at 4, 8 and 16 pounds/acre (active) to the row at time of planting resulted in peanut yields of 2275, 2554 and 2816 pounds/acre, respectively, compared with 787 pounds/acre for the nontreated controls. Acre values for the respective yields were $260.26, $303.67, $329.19 and $73.74, respectively. Broadcast surface applications of 16 pounds/acre (active) applied 6 days prior to planting resulted in a yield of 2633 pounds/acre valued at $295.95 compared with 1581 pounds/acre valued at $162.68 for the nontreated plots. In both row and broadcast treatments, reductions in nematode populations were directly correlated with the yield and acre value increases.

## INTRODUCTION

The sting nematode, Belonolaimus longicaudatus Rau, is a serious pathogen of peanuts, corn, cotton, soybeans and many other crop plants. In North Carolina greatest economic losses occur in the peanut belt where sting nematode is widespread. Because of the susceptibility to sting nematode of crops commonly grown in rotation with peanuts, considerable emphasis has been placed on chemical control. Extensive tests were conducted in 1958, 1959, and 1960 to evaluate the efficacy of various chemicals, formulations, dosages and time of application. Results of test treatments with fumigant type nematocides have been reported (2, 4). Results of the 1960 tests with O,O-diethyl O-2-pyrazinyl phosphorothioate (EN 18133) are presented here. A preliminary report has been given (1).

## MATERIALS AND METHODS

Location and Soil: These tests were conducted on the Barkeley Estate about 1 mile northeast of Severn, North Carolina, which had a history of poor yields of all crops for several years. Soybeans and corn grown in 1957 and 1958, respectively, and soybeans and peanuts in 1959, were severely affected by the sting nematode. The relatively uniform Norfolk loamy fine sand soil was heavily infested with sting nematode, but had very low populations of other plant-parasitic forms.

Soil Preparation: The debris of the preceding soybean and peanut crop was buried during March by turning the soil. The field was disked and harrowed just prior to initiating the study.

Plot Design and Treatments: The experimental design for row treatments was a randomized block with two-row (6 feet x 64 feet) plots, replicated eight times. Surface applications of a 10% granular formulation at 0, 4, 8 and 16 pounds/acre (active) were made on May 17, 1960 to a 12-inch band on the rows just prior to planting. Peanuts (N.C. 2 variety) were planted immediately following treatment with a 2-row tractor-drawn planter. The only mixing of nematocide into the soil was that incident with the planting operation.

Broadcast treatments consisted of eight 6-row plots (18 feet x 64 feet); four were treated with a 10% granular formulation at the rate of 16 pounds/acre (active) and four were nontreated. The chemical was applied to the soil surface with a Gandy spreader. It was mixed with the soil by disking once with a double gang disk harrow cutting 6 to 8 inches deep. Peanuts (N.C. 2 variety) were planted May 17, 1960, six days following treatment, with a 2-row planter. Subsequent cultural practices were essentially those recommended by the North Carolina Agricultural Extension service for peanut production in this area. Insecticides for foliage insects, however, were not applied as usage is dependent upon infestation.

Determination of Nematode Populations: An appropriate number of soil plugs 6 to 8 inches deep were taken from the root zone of plants in each plot with a 1-inch diameter soil sampling tube on June 27, August 18, and September 28, 1960. The nematodes were recovered from the soil by a combination of screens and modified Baermann funnel technique. Counts were made under a dissecting microscope (36X).

Harvest Operations: The peanut plants from each plot were removed on November 19, 1960 and stacked in the field on a pole. After drying until December 6 they were picked with a con-

ventional, cylinder type, peanut picker. The picked nuts from each plot were cleaned to remove the larger stem fragments before weighing. Samples of nuts for chemical residue analysis and market grade evaluation for each treatment were obtained by pooling a given quantity from each plot which received the same treatment.

## EXPERIMENTAL RESULTS

Yields: Results obtained with the row treatments relative to certain quality characteristics, price/cwt, yields and acre values are summarized in Table 1. The nematocide at all three rates significantly increased the yield of nuts in comparison with nontreated plots. Yields were significantly increased with each increase in dosage. Acre values are shown as the product of price/cwt and yields.

Table 1. Influence of soil treatments with EN 18133 for sting nematode control on peanut quality, yield and value per acre.

| Treatment (pounds/acre) | Fancy sized pods (%) | Extra large kernels (%) | Price/cwt. | Pounds/acre | Acre value (in dollars) |
|---|---|---|---|---|---|
| En 18133 4 | 27 | 28 | 11.44 | 2275** | 260.26 |
| 8 | 24 | 30 | 11.89 | 2554** | 303.67 |
| 16 | 21 | 23 | 11.69 | 2816** | 329.19 |
| Untreated check | 10 | 20 | 9.37 | 787 | 73.74 |
| L.S.D. at 5% | | | | 227 | |
| 1% | | | | 328 | |

Results obtained with broadcast treatments relative to certain quality characteristics and price/cwt were comparable to the 16 pounds/acre rate in the row treatments. Yields and acre value were 1581 versus 2633 and $162.68 versus $295.95 for nontreated and treated plots, respectively.

Nematode Control: The effects of the treatments on the number of nematodes per pint of soil for various sampling dates are shown in Table 2. The nematocide at each rate in row applications greatly reduced the nematode population below that found in the nontreated plots.

Table 2. Influence of soil treatments with EN 18133 on sting nematode populations in peanuts (1960).

| Treatment (pounds/acre) | Average no. sting nematodes/pint soil | | |
|---|---|---|---|
| | June 27 | Aug. 18 | Sept. 28 |
| Row Treatments | | | |
| EN 18133 4 | 62 | 94 | 75 |
| 8 | 72 | 50 | 40 |
| 16 | 75 | 25 | 40 |
| Untreated check | 144 | 181 | 38 |
| Broadcast Treatments | | | |
| EN 18133 16 | 31 | 0 | 38 |
| Untreated check | 150 | 144 | 75 |

These differences were evident for all dates except September 28. Differences between populations in plots treated with different rates were not very great; however, the population for the two higher rates was lower at the last two dates. Best control of the nematodes was obtained with the 16-pounds/acre rate of EN 18133 applied broadcast.

Residue Analysis: These studies are in progress.

## DISCUSSION AND CONCLUSIONS

The data presented show the value of reducing sting nematode population in fields where peanuts are planted. Improved quality as shown by market price in addition to increased yields resulted in substantial acre value increases for both row and broadcast treatments with EN 18133. These results are in close agreement with preliminary tests of the previous year (4) in which weighed quantities--4, 8 and 16 pounds/acre-- of EN 18133 was applied to the row by hand and mixed into the soil by rototilling twice. In this test EN 18133 at the 8-pounds/acre rate was comparable to the 1 gallon/acre granular of 1,2-dibromo-3-chloropropane (DBCP) in disease control, yield and quality evaluations.

At mid-season there was a noticeable difference in the amount of thrips, Frankliniella fusca (Hinds), and leafhopper, Empoasca fabae (Harr.), injury between treated and nontreated plots. Evidence that EN 18133 is systemic has been reported by Motsinger and Morgan (3). These investigators showed that soil applications of 1/2 and 1-pound/acre (active) of EN 18133 gave excellent control of an aphid, Myzus sp. It is probable that increased yield and quality of peanuts produced on treated plots in these tests was partially due to systemic control of these leaf feeding insects.

The broadcast application of the nematocide with a conventional fertilizer spreader and mixing into the soil with only a single disking represents one of the simplest methods of applying a nematocide. Simplicity of application, low phytotoxicity, as evidenced by the fact that planting can immediately follow treatment, all would indicate considerable promise for this material as a control for sting nematode and possibly leaf feeding insects on peanuts.

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# PRODUCTION OF "STERILE" ONION BULBS AND ROOTS FOR TESTING THE PATHOGENICITY OF FUNGUS ISOLATES

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The roots and stem plates of most field-grown onions contain Fusarium and bacteria internally and are, therefore, unsuitable for testing pathogenicity of other fungus isolates. Seedlings grown for inoculation work usually become contaminated before reaching a usable bulb size. Sterile seedlings produce roots too slowly and when inoculated produce symptoms typical of damping-off rather than bulb and basal rots. The use of field-grown bulbs for pathogenicity tests was a source of confusion to earlier workers (3, 4) and the problem of finding a suitable source of bulbs to inoculate for testing the pathogenicity of fungi isolated from diseased bulbs has continued.

Top set onions were tested and found to produce "sterile" bulbs (Fig. 1). The small bulbs produced on the seed stalk are not subject to penetration by soil organisms. When the outer scales were removed and the bulb was surface disinfested, these bulbs were nearly always free from fungi or bacteria that would grow on culture media. As the bulbs mature, or after a storage period, the outer scales tend to become infested with species of fungi such as Alternaria, Stemphylium, Penicillium and black Aspergillus.

Top set bulbs are produced in lieu of seed by certain varieties of onions. However, similar aerial bulb production can be induced on most varieties of onions by a process of clipping the developing seed stalk (1). This production of "head sets" on most varieties of onions makes it possible to use the "sterile" head set bulb to evaluate resistance of onion varieties to a single pathogen.

The bulbs are prepared for pathogenicity test by removal of the outer scales and disinfesting the surface of the top set bulb in 1:500 mercuric chloride in 50% ethyl alcohol for 5 minutes followed by three rinses in sterile water. These bulbs readily produce "sterile" roots following disinfestation.

FIGURE 1. "Sterile" roots and bulb produced from top set onions.

Following the water rinse the top set bulbs were placed on water agar, or between paper towels, or in greenhouse pots for root production. The inoculum was placed on the paper towel or agar plate near the stem plate for the pathogenicity test of the isolates. After allowing the fungus and roots to grow together for 5 to 7 days, several root segments were removed and examined for fungus penetration. The roots were surface disinfested in a sodium hypochloride solution of approximately 5000 ppm of available chlorine and incubated on nutrient agar.

Sterile seedling testing techniques have been developed by Gorenz, et al. (2) to evaluate onion resistance. The value of the seedling tests when compared with field results have been reduced because apparently many seedlings were eliminated which have resistance in the larger or bulb-sized plants and hence have valuable "field" resistance. With this "sterile" bulb production it is possible to test pathogenicity of single or multiple fungus isolates or to evaluate varietal resistance of bulb-sized onions to controlled infections without the complications of other unwanted infection, such as Fusarium and bacteria. With this sterile roots technique, several fungi isolated from field onions and not previously reported as parasitic were shown to be able to penetrate sterile onion roots.

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# LONG TERM STORAGE OF THE SUGARCANE MOSAIC VIRUS

Edwin H. Todd[1]

## Summary

Sugarcane mosaic virus in .01M solutions of sodium thiosulfate ($Na_2S_2O_3$) and sodium sulfite ($Na_2SO_3$), as reducing agents, was stored in vitro at -35° C for 12 months without any loss of infectivity. Temperature and light were considered important factors in evaluating infection rates during the peak summer months of July and August.

Mosaic of sugarcane, caused by a virus, nearly destroyed the sugarcane industry in Louisiana in the 1920's. As a result of strenuous research since that time, great advances were made in the production of resistant varieties which stabilized the sugarcane industry. Within the past few years, however, the apparent development of new strains of the mosaic virus and the release of susceptible varieties have again created a threat to the production of sugarcane in Louisiana (1), and in Florida as well. Since mosaic cannot be satisfactorily controlled by any known method of treatment, renewed efforts are being made toward the further development of resistant varieties (2).

In the search for commercially resistant varieties of sugarcane it is necessary to screen as many canes as possible for sources of resistance, and, subsequently, to test large progenies of crosses involving resistant parents. Such a procedure requires large quantities of inoculum at times when it may not be available in the field. Storage of the sugarcane mosaic virus, then, is especially important to sugarcane pathologists. Moreover, stored inoculum is valuable in that it relieves greenhouse space that ordinarily would be devoted to maintaining infected plants as sources of inoculum.

According to available literature, the longest period of sugarcane mosaic virus storage was 27 days (3, 4). Successful long term storage trials were conducted in 1959 at Canal Point, Florida with a mixture of four strains of the mosaic virus. The study was repeated in 1960 and is to be continued if the inoculum retains its infectivity.

## METHODS

The stored inoculum was prepared on December 29, 1959 at room temperature. Fresh, young expanded leaves and leaf spindles (excluding the growing points) of mosaic-infected sugarcane plants were coarsely chopped in a meat grinder. To each gram of chopped leaf material were added 3 ml of one of the following: distilled water, .01M sodium sulfite ($Na_2SO_3$), or .01M sodium thiosulfate ($Na_2S_2O_3$)[2]. These preparations were squeezed through cheese cloth into 250-ml Erlenmeyer flasks and test tubes. In order to allow for the expansion of the liquids during the freezing process, and thus avoid glassware breakage, only 100 ml of inoculum were put into the Erlenmeyer flasks; test tubes received 20 ml of the virus preparations. The containers were then plugged with cotton and immediately placed in a deep-freezer at -35° C. The calculated dilution factor for inoculum thus prepared was about 15.

The infectivity of stored and fresh mosaic inoculum was compared at monthly intervals, using a modification of the Matz inoculation technique (4). This procedure, which proved highly effective, consisted of applying a few drops of the inoculum in the leaf whorls of 2- to 3-week-old greenhouse plants of C. P. 31-294, a highly susceptible variety of sugarcane, and vigorously pricking through the inoculum into the young leaf tissues with a straight pin. All inoculations were conducted during the late morning hours.

## RESULTS

Data are shown in Table 1 for the key months only, although inoculations were conducted at monthly intervals.

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[1]Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

[2]Sodium sulfite and sodium thiosulfate were used primarily as reducing agents to prevent rapid oxidation.

Table 1. A comparison of the infectivity of freshly prepared and variously treated mosaic inoculum stored at -35° C, expressed as percentage infection of 10 to 20 inoculated plants.

| Treatment and dilution agent | Interval after preparation | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | 6 mos. (June) | fresh | 8 mos. (Aug.) | fresh | 10 mos. (Oct.) | fresh | 12 mos. (Dec.) | fresh |
| Stored inoculum: | | | | | | | | |
| Sodium thiosulfate solution .01M | 94 | | 66 | | 100 | | 92 | |
| Sodium sulfite solution .01M | 100 | | 75 | | 100 | | 92 | |
| Distilled water | 100 | | 25 | | 40 | | 25 | |
| Fresh inoculum: | | | | | | | | |
| Distilled water | | 100 | | 65 | | 90 | | 90 |
| Control | | 0 | | 0 | | 0 | | 0 |

## DISCUSSION

As indicated in Table 1, during the first 6 months no differences were recorded in the very high infection rates obtained with the fresh and stored inoculum. After 6 months, however, a steady decline was noted in the infectivity of stored inoculum diluted with distilled water. In addition, observations of results with this material indicated that a prolonged incubation period was required for symptom expression. The stored inoculum containing sodium thiosulfate and that containing sodium sulfite continued to show no loss of infectivity.

It is interesting to consider the sharp decline in the overall infection rates shown in Table 1 for the 8th month (August), and the subsequent return to very high infection rates shown for the 10th month (October). Actually, the decline first occurred in July and persisted at a comparable level in August as shown. Likewise, the return to high infection rates was first recorded in September, and they continued at the high level shown. A similar response was obtained for these months in the 1959 tests.

The source of inoculum, variety of sugarcane, size of plants, and time of day of inoculation were consistent throughout the test. Insofar as can be determined, temperature and light were the only uncontrolled environmental factors involved in this study. It is believed, therefore, that higher temperatures and/or photoperiod, affecting the physiology of the plants, may have been critical factors responsible for the lower infection rates obtained during the months of July and August.

In view of the data shown it is apparent that the sugarcane mosaic virus can be successfully stored in vitro at -35° C for at least 12 months without loss of infectivity, provided a reducing agent is added to the inoculum to prevent rapid oxidation. Also, it appears that temperature and/or light, as they affect physiological processes of the plant, may be important factors to consider in evaluating results obtained in mechanical inoculation tests with the sugarcane mosaic virus.

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# PREVENTION OF BARK BEETLE DEVELOPMENT IN UNDESIRABLE ELMS FOR THE CONTROL OF DUTCH ELM DISEASE

E. B. Himelick and Dan Neely[1]

## Abstract

Thirty-six chemicals were introduced into the sap stream of living American elm trees to determine their effectiveness in preventing elm bark beetle development. Of the chemicals tested, only sodium arsenite when applied in ax frills gave practical and virtually complete control. From field studies conducted on large elms in three different forested areas in northern Illinois, over 97% control was obtained when diseased trees were treated during the incipient stage of wilt development. No elm bark beetles were found in trees that were without disease symptoms at the time of treatment.

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Most municipalities ignore the diseased trees in elm populations occurring outside their residential and business areas. The occurrence of stands of wild elms along rivers and streams and in wooded areas is a major problem to many cities that are attempting community-wide control of Dutch elm disease by sanitation procedures. Elms affected with the Dutch elm disease in these wild stands provide a source of inoculum of the causal fungus, Ceratocystis ulmi (Buis.) Moreau, and also provide a habitat suitable for breeding and population increases of the two bark beetle vectors, Scolytus multistriatus (Marsh.) and Hylurgopinus rufipes (Eich.). The present methods of controlling the disease, sanitation and spraying, are not practical procedures to be used in wooded and forested areas.

Craighead and St. George (2) showed that certain chemicals when introduced into the sap stream killed trees and also prevented bark beetle breeding in the treated trees. Whitten (7) reported more comprehensive experiments involving additional chemicals, aimed specifically at killing elm trees to control the two insect vectors of Dutch elm disease. Whitten used a watertight collar of rubberized fabric as described by Lantz (4) to introduce solutions into healthy elms. Water solutions of sodium arsenite, copper sulfate, ammonium bifluoride, zinc chloride, copper nitrate and copper chloride were effective in killing elm trees and preventing sprouting of the roots.

Investigators (1, 8) have shown that sodium arsenite is an effective silvicide and it is now used routinely as a debarking compound in many forests. During experiments on the control of oak wilt, Himelick (3) noted that when applied in a complete ax frill at the base of the tree, sodium arsenite was carried throughout the oak tree in concentrations sufficiently high to limit bark borer development for a period of at least 1 year. Experiments in 1957 and 1958 were designed to find out whether solutions of this chemical, when applied in a similar manner, would work equally well in elms to prevent elm bark beetle development.

## PRELIMINARY STUDIES

Methods of injecting chemicals into the sap stream of trees are well described by May (5). Several of these methods were used in the present study to introduce solutions or salts into elms. Watertight collars were prepared by completely girdling the tree to a depth of 3/4 inch into the wood with a chain saw. A beveled cut was made with a wood chisel through the bark and wood down to the girdle. Caulking compound was applied to the base of the cut and heavy plastic sheeting was wrapped around the tree so that the lower edge of the sheeting extended just below the girdle. The sheeting was pulled tightly into the girdle and bound with heavy cord. Chemical solutions were then poured into the collar. In other trees solutions or salts were placed in regularly spaced holes prepared either with a poison ax or a 3/4-inch bit in an electric drill. These holes were plugged flush with the cambium with rubber stoppers. Other trees were girdled with a gasoline engine-powered "Little Beaver," or girdled by making complete ax frills close to the base of the trees to be treated. The wood was cut to a depth of at least 1/2 inch in a complete girdle in both instances.

After 56 elms ranging from 13 to 24 inches dbh had been treated with several different chemicals during 1957 and 1958, it was evident that the ax frill method was the only effective

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and practical way of treating large numbers of trees. Our observations on elms agreed with those of Craighead and St. George (2) that "inasmuch as there is little lateral movement of the sap stream within the stem of a tree, a chemical introduced at one point will go up the stem in only a narrow strip. Therefore, it is necessary to introduce the materials around the entire circumference of the tree in order to get adequate and uniform distribution to all parts of the stem and crown." The plastic collar, though effective, required considerable time and effort to construct. The "Little Beaver" girdler made a rounded cut that did not hold a sufficient quantity of liquid chemical for uptake by the vessels of the wood.

During June and July in 1957 and 1958, 30 American elm trees affected with Dutch elm disease in varying stages of wilt were treated with sodium arsenite[2]. The ax frill method of application was used. The undiluted chemical was applied with a 2 1/2-gallon sprayer. The spray nozzle was removed from the end of the spray tube to allow dispensing of the liquid at low pressure. The person dispensing the chemical circled the tree three times, allowing the chemical to run slowly into the frill. Use of low pressure and careful dispensing of the chemical resulted in very little runoff.

The results obtained from the use of sodium arsenite varied with the stage of wilt at the time of treatment. In trees that were already defoliated by Dutch elm disease when treated in 1957 and 1958, there was not sufficient movement of sodium arsenite to prevent bark beetle attack on either the trunk or the branches in the crown. However, trees treated when 20 to 50% of the foliage was wilted had trunks free of living bark beetles, and bark beetle galleries were present only in those branches that had exhibited late stages of wilt. Those trees treated during early wilt symptoms (3 to 10% of the crown) were completely free of living elm bark beetles except on the one or two branches containing the fungus pathogen. No elm bark beetles were found in those trees without disease symptoms at the time of treatment.

Because of the high mammalian toxicity of sodium arsenite, a screening program was conducted on 36 miscellaneous chemicals to determine if another less toxic compound would be as effective in preventing bark beetle attack. The chemicals were tested on healthy elms 4 to 8 inches dbh during June and July of 1959. They were applied by slowly pouring 300 to 500 ml quantities into an ax frill that had been cut 2 feet above the ground line. The effectiveness as silvicides and the control of bark beetle development for the 36 chemicals used in this test are given in Table 1. Those chemicals that showed the most promise in the screening test were then applied on larger elm trees (15 to 20 inches dbh) on July 30, 1959. Each of seven materials, Acti-dione, ammonium bifluoride, copper chloride, copper nitrate, sodium arsenate, sodium chlorate, and Vapam, was applied to two or more trees. None of these chemicals was considered effective in preventing bark beetle attack when applied by the ax frill method.

To aid in estimating the percentage reduction in the bark beetle population emerging from elms poisoned with sodium arsenite, the bark area of trees of measured diameters was determined. Trees to be measured were selected on woodland and farm areas in northern Illinois. Branches smaller than 1 inch in diameter were not included in the determinations since few elm bark beetles breed in such small branches. Our determinations of tree surface areas compare closely with those obtained by Wolfenbarger (9). A line graph of measurements taken from 34 elms in our studies is shown in Figure 1. Since a branch has a surface area roughly equal to that of a tree with the same diameter, it is possible to calculate the percentage of bark area of a branch of any size on a particular tree.

## FIELD STUDIES

Since sodium arsenite tested best in effectively preventing bark beetle attack, the chemical was tested on a larger scale on trees in forested and wooded areas in northern Illinois. In Sinnissippi Forest near Oregon, Illinois 22 American elms 13 to 24 inches dbh and having varying degrees of wilt symptoms were used. The trees were treated with a commercially prepared sodium arsenite in ax frills during July 1958. Care was taken to prevent excessive runoff. The stage of wilt in each tree was recorded at the time of treatment. Readings on bark beetle development in the treated trees were made on November 6 and 7, 1958.

Elms with Dutch elm disease that occur naturally in wooded areas along rivers and streams are a major problem to neighboring cities practicing disease control procedures. To assist the Dutch elm disease sanitation efforts of Batavia, a city located along the Fox River in Illinois, we treated 19 elms growing in ravines and along the river with Atlas D formulation

[2]Atlas D formulation sold by the Chipman Chemical Company as a debarking compound, contains 40% active sodium arsenite, a blue dye, and a deer repellent.

Table 1. The effectiveness of chemicals introduced into the sap stream of living elms in preventing bark beetle development.

| Chemical | Concentration | Effectiveness in killing elm trees | Effectiveness in preventing bark beetle development |
|---|---|---|---|
| Acetic acid | concentrated | poor | poor |
| Acetone | 100% | poor | poor |
| Acti-dione[a] | 10 g/l | fair | poor |
| Aero cyanate[b] | 100 g/l | poor | poor |
| Amino-triazole | 100 g/l | fair | poor |
| Ammate[c] | 100 g/l | poor | poor |
| Ammonium bifluoride | 500 g/l | fair | poor |
| Borax[d] | 100 g/l | poor | poor |
| Copper chloride | 200 g/l | good | good |
| Copper napthanate | concentrated | poor | poor |
| Copper nitrate | 200 g/l | good | fair |
| Copper sulfate | 100 g/l | good | fair |
| Ethyl alcohol, 95% | stock concentration | poor | poor |
| Fluoroboric acid | 5% | poor | poor |
| Formalin | stock concentration | poor | poor |
| Hydrochloric acid | concentrated | good | poor |
| Organic mercury fungicides (Tag, Phix, Coromerc, and Puratized Agricultural Spray[e]) | stock concentration | poor | poor |
| Naphthalene | 40 g/l ethanol | poor | poor |
| Potassium hydroxide | 100 g/l | poor | poor |
| Potassium permanganate | 80 g/l | poor | poor |
| Sevin[f] | 53 g/l | poor | poor |
| Sodium arsenate | 100 g/l | excellent | good |
| Sodium arsenite | 40 g/l | excellent | excellent |
| Sodium chlorate | 600 g/l | good | poor |
| Sodium chloride | 120 g/l | poor | poor |
| Sodium hydroxide | 200 g/l | poor | poor |
| Sodium hypochlorite | 5.35% | poor | poor |
| Sodium pentachlorophenate | 100 g/l | poor | poor |
| Sodium fluoride | 50 g/l | poor | poor |
| Sulfuric acid | concentrated | fair | poor |
| Vapam[g] | diluted 1 to 3 | good | poor |
| Zinc chloride | 200 g/l | fair | poor |
| 2,4,5-T[h] | 2% in fuel oil | good | poor |

[a]$\beta$-(2-(3,5-dimethyl-2-oxocyclohexyl)-2-hydroxyethyl)-glutarimide. [b]potassium cyanate. [c]ammonium sulfamate. [d]sodium tetraborate. [e]Tag and Phix = phenylmercury acetate; Coromerc = N-phenylmercuriethylenediamine; Puratized Agricultural Spray = phenylmercuri triethanol ammonium lactate. [f]1-naphthyl methylcarbamate. [g]sodium n-methyl dithiocarbamate. [h]2,4,5-trichlorophenoxyacetic acid.

of sodium arsenite in ax frills on June 16, 1959. The majority of the trees selected showed incipient stages of Dutch elm disease. Again care was taken to avoid runoff of the chemical. Following application of the arsenite, the frill and the basal portion of the trunk were thoroughly sprayed with creosote to prevent animal feeding.

To investigate the usefulness of poisoning infected trees in a forested area under intensive management, tests were conducted in 1959 and 1960 in the more remote areas of the Cook County Forest Preserve with the cooperation of personnel of the Forest Preserve District[3]. The number of cases of Dutch elm disease in the preserve, which contains 46,000 acres, has increased from six infected elms in 1955 to more than 7000 discovered in 1960. The total loss to date (less than 10,000 elms) is only a little more than 1/2 of 1% of the total estimated elm population. The labor crew of the Forest Preserve District scouted the areas for diseased

[3]Acknowledgment and thanks are especially due Mr. Noel B. Wysong and Mr. James Tyndall, Chief Forester and Forester respectively, Cook County Forest Preserve, River Forest, Illinois.

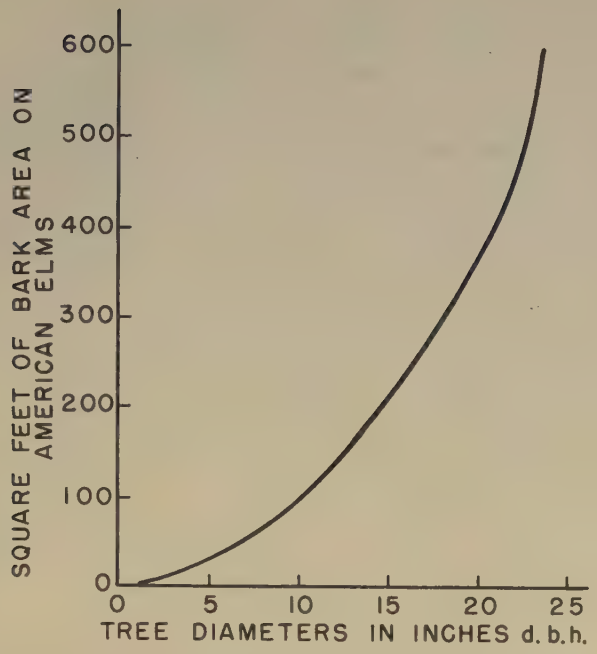


FIGURE 1. The bark area on American elm trees of given diameters measured at breast height.

Table 2. The effectiveness of sodium arsenite applied in ax frills in preventing the attack and development of the two elm bark beetles in diseased elms.

| Location | Wilt development at time of treatment | Number of trees | Average dbh | Total bark area (sq. ft.) | Bark area infested with beetles (sq. ft.) | % control[a] |
|---|---|---|---|---|---|---|
| Sinnissippi Forest | incipient | 6 | 19.0 | 2106 | 61 | 97.1 |
| | early | 2 | 25.5 | 1015 | 133 | 86.9 |
| | advanced | 14 | 18.9 | 4640 | 3600 | 22.4 |
| Batavia | incipient | 15 | 17.5 | 4840 | 28 | 99.4 |
| | early | 1 | 14.0 | 190 | 35 | 81.6 |
| | advanced | 3 | 15.7 | 709 | 449 | 36.7 |
| Busse Forest | incipient | 28 | 15.4 | 8728 | 125 | 98.6 |
| | early | 30 | 17.5 | 10554 | 2318 | 78.0 |
| | advanced | 25 | 14.5 | 5764 | 3750 | 34.9 |

[a]% of total bark area not infested with bark beetles.

trees, treated the trees with sodium arsenite, and applied the creosote. Notes on tree location, tree size, and stage of wilt were recorded at the time of treatment. In 1959, 100 elms were treated during the last week of July. Treatment of trees this late in the season had allowed considerable time for disease progress during the early summer. In 1960, over 2000 elms were treated in June, July, and August. Thirty-two of the elms treated in 1959 were selected at random from those treated in Busse Forest, were cut during December, and were carefully examined for the presence of elm bark beetles. A random sample of the elms treated during June, July and August 1960 was cut and examined in December 1960.

The results from using sodium arsenite on large diseased elms in the three areas of northern Illinois are shown in Table 2. The total bark area of the trees and the bark area on the branches infested with beetles were obtained by using the line graph (Fig. 1). From 97.1 to 99.4% of the bark area on those elms showing incipient wilt at the time of treatment was not infested by either the American or European elm bark beetle. When wilt symptoms had progressed to the point that branches 5 to 8 inches in diameter were defoliated at the time of treatment, 78.0 to 86.9% of the surface area of the trees remained free of beetles. Considerably less control (22.4 to 36.7%) was obtained when trees were treated in the advanced stage of symptom expression of the Dutch elm disease.

## DISCUSSION

Whitten (7) demonstrated that sodium arsenite, when introduced internally into elms by means of a watertight collar, was effective in killing elm trees and in preventing bark beetle attack and development. This information has not been used or integrated into a Dutch elm disease sanitation program for two reasons: 1) sodium arsenite is poisonous to humans, both orally and by contact, and 2) application of the chemical by the preparation of watertight collars around each elm to be treated is too costly and time consuming to be practical.

After making an intensive study on the effect of chemical debarking on forest wildlife, Webb, Rosasco, and Simkins (6) showed that there is no indication that the loss of wildlife is significant if sodium arsenite is carefully handled and dispensed. They suggested the addition of a repellent to the debarking chemical. Sodium arsenite with the addition of cresylic acid and creosote derivative repellents is commercially available as Herbicidol DB, Atlas C, and Atlas D. The use of arsenic compounds containing repellents and dyes, and the use of an additional creosote spray applied to the ax frill and tree trunk following application of the arsenic, should greatly reduce, if not eliminate, the possibility of animal contact with the poison. Regardless of safeguards, sodium arsenite should not be used in populous areas.

Although the ax frill allows the introduction of only limited amounts of chemical into the sap stream of elms, the method was completely effective in killing elms as large as 36 inches dbh without rendering them liable to elm bark beetle attack. Of all methods tried for the introduction of chemicals into the sap stream, the ax frill is the most practical method for foresters. It requires only an ax and two sprayers (one for sodium arsenite and the other for creosote) to be carried into the forest.

Best control of the elm bark beetle populations can be accomplished by poisoning all undesirable elms while they are still healthy, and poisoning other elms as soon as the initial Dutch elm disease symptoms become evident. Sodium arsenite placed in properly made frills on healthy elms prevents all bark beetle development in the treated trees. Previous mechanical injuries or an unusual formation of the wood grain may cause an occasional skip in the distribution of the chemical, thereby allowing a few beetles to mature. Treatment of diseased elms showing wilt symptoms on branches less than 4 inches in diameter has rendered 97.1 to 99.4% of the bark of these trees unsuitable for bark beetle development. The chemical moves in the vessels throughout the trunk and into all branches not completely plugged by the action of Ceratocystis ulmi. The presence of sodium arsenite prevents elm bark beetle development in areas where plugging has not yet occurred.

The present Dutch elm disease control practice in wooded areas of cutting and burning diseased trees is extremely costly in terms of both manpower and machinery. The cost of sanitation practices in such areas would be greatly reduced by use of sodium arsenite. Over 2000 elms were treated with sodium arsenite in the Cook County Forest Preserve in 1960 at a saving of $30.00 per tree. The annual incidence of Dutch elm disease in the preserve is expected to soon reach 20,000 cases. The cost of the conventional sanitation measures of destroying elm bark beetle breeding material will then become prohibitive.

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# SEED TRANSMISSION OF COFFEE RING SPOT BY EXCELSA COFFEE (COFFEA EXCELSA)

Tiburcio T. Reyes[1]

Seed transmissions have been demonstrated to occur in a number of virus diseases in plants but apparently there have been no reports of those affecting coffee. Ring spot disease of coffee, caused by a virus, was reported in Brazil (1), Costa Rica (3), and in the Philippines (2). The virus is easily transmitted by grafting and by the aphid Toxoptera aurantii (Fonsc.).

In the middle of March 1959 a survey of the distribution of the disease in the coffee plantation of the College of Agriculture and Central Experiment Station was conducted. In this survey cases of natural infection on Excelsa seedlings were found under randomly distributed ring spot-affected trees. Not a single case was observed on Arabica seedlings. The symptoms of the disease on seedlings compared favorably with those on older plants. Several of these infected seedlings were brought to the Plant Pathology greenhouse for transmission studies. Of course, the fact that ring spot infection occurs naturally on seedlings does not necessarily mean that it is transmitted through the seed, but the writer believes that it is a strong circumstantial evidence to that effect. Greenhouse tests were undertaken to determine whether coffee ring spot is transmitted through the seed of Excelsa coffee.

Approximately 100 ripe fruits were collected from a severely affected Excelsa tree in the coffee plantation of the College of Agriculture and Central Experiment Station on March 17, 1959. The seeds obtained from these fruits were sown in two pots of sterilized soil in the greenhouse. On March 28, 1959, 60 more ripe fruits were collected from the same diseased Excelsa tree. The seeds were sown in a pot of sterilized soil in the greenhouse. Two pots were also sown with seed collected from a healthy Excelsa tree. The developed seedlings from all plantings were examined for ring spot infection at least twice a month. All experimental and non-experimental plants in the greenhouse were sprayed monthly or bimonthly with S-(1,2-bis(ethoxycarbonyl)ethyl)O,O-dimethyl phosphorodithioate (malathion) against insects.

On October 18, 1960, a total of four plants, or 7.1%, of the 56 seedlings resulting from the first planting exhibited typical ring spot symptoms; of the 47 seedlings from the second planting a total of five plants, or 10.6%, showed the disease. The first plants to show signs of virus symptoms were noted on September 3, 1960. No additional infection was recorded on January 7, 1961, when the seedlings were last examined. None of the 51 controls grown from seed from healthy plants showed the disease. Although mealy bugs and scale insects were found on some of the non-experimental plants none were observed on the experimental ones.

From August to October 1959, numerous seeds obtained from ring spot-affected Arabica trees were planted in seed flats and in small isolated plots. No symptoms developed in any of the 562 plants obtained from such seed after 15 to 18 months. These results corroborate the writer's (2) previous conclusion that the disease was probably not seedborne. Wellman (3) obtained similar result in his experiment on seed transmission of blister spot of Arabica coffee from virus in Costa Rica. If the disease is carried through the seed of Arabica, probably a much longer incubation period is required for development of symptoms.

These tests and field observations seem to prove the transmission of coffee ring spot through the seed of Excelsa but not of Arabica. Excelsa seed has probably played a part in the distribution of ring spot. The limited and erratic distribution of the disease in the College plantation could perhaps be explained by the fact that seedlings showing mild symptoms and mistaken for early rust infection were not discarded at the time of planting. These diseased seedlings had perhaps been planted at random.

Experiments on the transmission of the disease by seeds of Liberica and Robusta coffees and by insects are in progress.

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# POLYPORUS SPP. ASSOCIATED WITH WOOD DECAY OF LIVING PEACH TREES IN SOUTH CAROLINA[1]

D. H. Petersen[2]

## Summary

Thirteen species of Polyporus were found associated with decayed woody tissues of living peach trees in South Carolina. Polyporus curtisii, P. gilvus, P. hirsutus, P. palustris, P. pargamenus, P. tulipiferus and P. versicolor were found most frequently and with the exception of P. pargamenus appeared to be agents of extensive damage to living peach trees. The following species are reported apparently for the first time on peach in the United States: P. adustus, P. albellus, P. arcularius, P. dichrous, P. palustris, P. sanguineus and P. sulphureus.

## INTRODUCTION

One cause of rapid decline of many peach trees in the southeastern United States appears to be the decay of sapwood and heartwood by various species of Hymenomycetes. Petersen (7) reported 22 species of this group excluding Polyporus spp. as associated with decay of woody tissues of living peach trees. This paper reports the results of a survey of Polyporus spp. occurring on peach[3]. The procedures used in the survey were the same as those used previously (7).

## RESULTS AND DISCUSSION

A brief discussion of each species follows. Table 1 indicates the general frequency of the various species.

Table 1. Frequency of occurrence of Polyporus spp. on living peach trees in South Carolina.

| Species | Frequency of occurrence[a] | Species | Frequency of occurrence[a] |
|---|---|---|---|
| P. adustus | 1 | P. palustris | 4 |
| P. albellus | 1 | P. pargamenus | 3 |
| P. arcularius | 2 | P. sanguineus | 3 |
| P. curtisii | 4 | P. sulphureus | 1 |
| P. dichrous | 1 | P. tulipiferus | 4 |
| P. gilvus | 4 | P. versicolor | 4 |
| P. hirsutus | 4 | | |

[a] Symbols for the numbers of times found: less than 10 -- 1; 10-25 -- 2; 26-100--3; 101-1000--4.

Polyporus adustus Willd. ex Fr. was seen rarely on peach trees. The sporophores were collected from dead tissue of living trunks and were associated with a white, mottled rot of the sapwood. No reference to the occurrence of this species on peach was found.

Polyporus albellus Pk., seen rarely, was found on dead tissues of limbs. The sporophores were associated with a white, soft rot which was extensive in both sapwood and heartwood. No reference to P. albellus on peach was found. However, P. lacteus Fr. was reported as causing a sapwood rot of peach in North Carolina (9) and Overholts (6, p. 299) indicated that most collections of P. lacteus belong under P. albellus.

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[1] Cooperative investigations of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture and the South Carolina Agricultural Experiment Station.

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[3] Acknowledgment is given to J. L. Lowe, New York State College of Forestry, Syracuse, New York, Beltsville Forest Disease Laboratory, Forest Service, J. A. Stevenson and P. L. Lentz, Crops Research Division, Agricultural Research Service, Beltsville, Maryland, for aid in identification.

Polyporus arcularius Batsch ex Fr. was found on peach trees only in the northern part of South Carolina. Its sporophores were collected from dead areas of limbs and trunks in regions apparently damaged by sun-scald or winter injury. They were associated with a firm, yellowish-white decay of both sapwood and heartwood. The area of decay was not extensive. No reference to its association with peach was found.

Polyporus curtisii Berk. (Ganoderma curtisii (Berk.) Murr.) was found frequently on living peach trees throughout the State. Its sporophores were located usually at or near the soil line on the trunk or on the soil nearby. In the latter instances, the base of the stipe was attached to an infected root beneath the soil surface. The species was found on apparently healthy trees, on trees in various stages of decline, and on those that had died recently. No sporophores were found on trees which had been dead for more than a year. P. curtisii appeared to cause extensive decay of the larger roots and underground trunk portions of the trees. Decayed tissues were soft, white, and soggy.

Under the name Ganoderma curtisii, the species was reported on peach in North Carolina and Virginia (9). Rhoads (8) reported it in South Carolina and Georgia. The author, observing P. curtisii in orchards for a period of 10 years, has seen trees die in ever-widening circles beginning from two or three infected trees. These observations indicate that a study of the relationship of this species to peach tree decline is warranted.

The infrequent sporophores of Polyporus dichrous Fr. were seen on dead tissue of living limbs and trunks. The associated decay was very soft, punky, and light straw-colored. Oxidase tests on this species were negative (2, 5), indicating the decay to be a brown rot. Overholts (6, p. 363) expressed the opinion that the decay in various hardwood species was of the brown decarbonizing type, but that it progressed so slowly it became surrounded by decay caused by other fungal species. Observations in South Carolina orchards indicated that P. dichrous was not important in wood decay of living peach trees. No reference was found to its association with peach.

Polyporus gilvus (Schw.) Fr. was associated very commonly with peach wood decay in South Carolina orchards. The fruiting bodies were seen on tree trunks from the soil line to the crotch area and occasionally near the base of large scaffold limbs. The fungus appeared to persist in the trunks and limbs for only a few months after their death. Hirt (4) was of the opinion that the hyphae of this fungus could subsist in wood tissues only when they were killed by other means. Field observations and isolations by the author, however, indicate that P. gilvus probably causes death of tissues in advance of hyphal growth and the fungus is regarded as one of the major species causing peach wood decay in South Carolina. The sporophores were associated with an extensive white rot of the sapwood and heartwood in which the wood became porous, flesh-colored, and brittle when dry. Rhoads (8) reported P. gilvus as attacking peach limbs and trunks in South Carolina and Georgia.

Polyporus hirsutus Wulf. ex Fr. has a cosmopolitan distribution on peach in the United States (9). It was one of the most common species of Hymenomycetes found in South Carolina orchards. Fruiting bodies were seen on trunks and on limbs over 2 years old. The fungus was associated with a white decay of sapwood and heartwood in which the tissues finally became light and spongy. P. hirsutus fruited on trees or parts of them for several seasons following tree death. Weir (10), using the name Polystictus hirsutus, reported that this species caused decay of sapwood and heartwood in living peach trees in Idaho, Washington, Oregon, and Indiana. He stated that the fungus entered the trees through pruning wounds, frost checks, and wounds made by cultivation equipment. From orchard observations in South Carolina it appears that this species may play a prominent role in wood decay of peach trees.

Polyporus palustris Berk. & Curt. was found repeatedly associated with the decay of living peach trees in South Carolina. During the study it was also observed commonly in orchards in Georgia and North Carolina. Penetration of the wood appeared to be primarily through pruning wounds. The decay was a brown cubical rot in which sheets of mycelium were readily evident between the cubes of decayed wood. Rapid destruction of the heartwood often resulted in the collapse of limbs under the weight of growing fruit before there was any external evidence of infection.

P. palustris was reported only as a saprophyte on the dead wood of conifers (6, p. 281), although it appears likely that P. submurinus, which Rhoads (8) reported on peach in South Carolina and Georgia, may be P. palustris.

Based on the frequency of its occurrence and the extensive decay associated with its fruiting bodies, P. palustris may well be the most destructive Hymenomycete on peach in South Carolina where it appears annually to cause much loss of fruit production. Studies of the fungus and its possible associates, its biology, pathogenicity and control are underway.

Polyporus pargamenus Fr. was found commonly fruiting on dead tissue of limbs and trunks throughout South Carolina. The fruiting bodies were associated with a soft, spongy, flesh- to straw-colored rot mostly of the sapwood. Elliott (3), using the name Coriolus prolificans, termed the fungus the cause of a "severe parasitic wood-rot" of peach trees in Arkansas when trees were pruned so as to expose the heartwood. In South Carolina the fungus does not appear to be a major factor in peach wood decay.

Polyporus sanguineus L. ex Fr. was found commonly in northern South Carolina, but rarely in the remainder of the State. Most sporophores were associated with dead tissues of living limbs, but occasionally they were found on dead limbs of living trees and dead tissues of living trunks. The fruiting bodies were associated with a soft, white rot of the sapwood and heartwood. No literature reference was found associating P. sanguineus with living peach trees.

Polyporus sulphureus Bull. ex Fr. was seen in the crotch area of two living trees. The associated decay was an extensive, brown, cubical rot, the cracks of which were filled with leathery, yellow-brown mycelium. Because of its infrequent occurrence, this species is not considered a major factor in the decay of living peach trees in the State. This appears to be the first report of association of P. sulphureus with living peach trees.

Polyporus tulipiferus (Schw.) Overh. was one of the most common Hymenomycetous species associated with decay of living trees. It occurred throughout the State on all aboveground woody portions of the tree. The decay, primarily of the sapwood, was a white, soft rot. Because of its frequent occurrence on trees of all ages, P. tulipiferus may play a major role in the decay of peach wood tissues.

Rhoads (8) found P. tulipiferus on peach in South Carolina and Georgia. This fungus undoubtedly has a much wider distribution on peach, but there has been some confusion in its nomenclature. According to Overholts (6, p. 329), Irpex lacteus Fr. is a synonym. Since many authors do not consider Irpex a valid genus, the fungus would become Polyporus lacteus or Polystictus lacteus. In his discussion of Polyporus albellus Pk., Overholts (6, p. 299) stated that most American collections of P. chioneus and P. lacteus belong under P. albellus. Thus, it is uncertain to which of the two species, Polyporus tulipiferus or P. albellus, the references of P. lacteus on peach belong, since both species, in the sense of Overholts, have been observed on peach in South Carolina. Wilson (11) reported P. tulipiferus as parasitic on peach in Iowa and Weir (10) stated that Polystictus lacteus caused a sapwood and heartwood rot of living peach trees. Overholts (6, p. 331) referred the fungus in both of these references to P. tulipiferus.

Polyporus versicolor L. ex Fr. was found very commonly throughout South Carolina attacking woody parts of living trees, including the crown below the soil line. The decay of the crown tissues could not be distinguished from that caused by Clitocybe tabescens without resorting to characteristics in pure culture. The decay caused by P. versicolor was a white to straw-colored, spongy rot of the sapwood and, to a limited extent, the heartwood.

The fungus is reported as of cosmopolitan distribution in the United States as a cause of peach sapwood rot (9). Elliott (3), using the name Coriolus versicolor, termed it a parasite on peach in Arkansas. Birmingham (1), using the name Polystictus versicolor, described infection as resulting in the death of bark and wood in Australia and termed the disease "yellowish wood rot." Observations made during this study indicated the fungus may be a major cause of decay in living peach trees.

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# STUDIES OF THE HOST RANGE OF MELOIDOGYNE INCOGNITA ACRITA[1]

H. W. Crittenden[2]

This report covers field and greenhouse studies in Delaware of the host range of Meloidogyne incognita acrita Chitwood, 1949. An earlier report by Gaskin and Crittenden[3] covered a host range for M. hapla in Delaware. These are the two species of root-knot nematodes commonly found associated with plants in Delaware.

## METHODS

Tests of plant reaction to M. incognita acrita were conducted in the field at Bethel, Delaware. Each variety of plant was grown in four replications in heavily infested soil for 100 days. The tests were conducted under environmental conditions that were conducive for females to deposit eggs in 40 days. The soil was removed from the test plants by carefully washing the roots in water. A total of 40 plants of each variety was examined macroscopically in the field for the presence of root-knot galls on the roots.

The same varieties of plants tested in the field were also tested for their reaction to M. incognita acrita in the greenhouse at Newark, Delaware. The soil for tests in the greenhouse was prepared by using a loamy sand and incorporating chopped, heavily galled roots obtained from Bethel, Delaware. Test plants were grown in the infested soil for at least 60 days, then soil was removed from the roots with water and the roots examined under a binocular dissecting microscope for the occurrence of female nematodes and egg masses.

The reaction ratings used were as follows: 1--no nematodes in the roots; 2--only a few adult females in the roots; 3--a moderate number of females in the roots; 4--many females in the roots; and 5--an extremely high number of females in the roots. A variety of a plant species was considered susceptible (marked S) when the roots were classified in classes 2, 3, 4, or 5. A variety was considered resistant (marked R) only when the average of numerous observations of females in the roots fell between classes 1 and 2. The resistant varieties are classified from 1.0 to 1.9, depending upon the degree of resistance.

## RESULTS AND DISCUSSION

The results, reported in Table 1, were similar both in the field and in the greenhouse. The reaction rating is a composite figure for a variety in both locations.

The fact that resistance to M. incognita acrita exists in soybean (Glycine max) has enabled a breeding and selection program, for improved soybean varieties with resistance to this species of nematode, to proceed in Delaware and other areas.

The fact that resistance to M. incognita acrita exists in oat (Avena sativa) has been used in a control recommendation, namely, that a resistant oat variety should be used as a winter cover crop on soils in southern Delaware that are infested with this nematode.

The combination of the use of a resistant soybean variety in summer with a resistant oat variety as a winter cover crop has a high value for nematode control.

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[3]Gaskin, Timothy A., and H. W. Crittenden. 1956. Studies of the host range of Meloidogyne hapla. Plant Disease Reptr. 40: 265-270.

Table 1. Reaction of plant varieties to Meloidogyne incognita acrita.

| Scientific name | Common name | Variety | Reaction rating |
|---|---|---|---|
| Gramineae | | | |
| Avena sativa | oat | Arkwin | S(4) |
| | | Arlington | R(1.3) |
| | | Atlantic | R(1.4) |
| | | Coy | R(1.8) |
| | | DeSoto | R(1.4) |
| | | Dubois | S(3) |
| | | Forkedeer | S(4) |
| | | Frazier | R(1.6) |
| | | Fulghum | R(1.8) |
| | | Fulwin | S(3) |
| | | Leconte | S(3) |
| | | Lee | S(3) |
| | | Lemont Cross | R(1.3) |
| | | Letoria | S(4) |
| | | Mustang | R(1.4) |
| | | Stanton | S(4) |
| | | Traveler | S(4) |
| | | Victorgrain | R(1.5) |
| | | Victoria | R(1.6) |
| Hordeum vulgare | barley | Colonial | S(3) |
| | | Fayette | S(4) |
| | | Harbine | S(3) |
| | | Kentucky 1 | S(3) |
| | | Piedmont | S(3) |
| | | Texan | S(4) |
| | | Wong | S(4) |
| Secale cereale | rye | Albruzzi | S(2) |
| | | Balbo | S(2) |
| | | Imperial | S(2) |
| Triticum aestivum | wheat | Butler | S(4) |
| | | Nittany | S(4) |
| | | Nudel | S(3) |
| | | Nured | S(4) |
| | | Pennol | S(4) |
| | | Redhart | S(4) |
| | | Seneca | S(4) |
| | | Thorne | S(4) |
| Liliaceae | | | |
| Asparagus officinalis | asparagus | Martha Washington | R(1.2) |
| Leguminosae | | | |
| Glycine max | soybean | Adams | S(5) |
| | | Aoda | S(3) |
| | | Arksoy | S(4) |
| | | Avoyelles | S(4) |
| | | Barchet | S(4) |
| | | Biloxi | S(4) |
| | | Blackhawk | R(1.5) |
| | | Cayuga | S(4) |
| | | Charlee | S(4) |
| | | Cherokee | S(4) |
| | | Chief | S(3) |
| | | Clark | S(3) |
| | | CNS | S(4) |
| | | Creole | S(4) |
| | | Delsoy | S(3) |
| | | Delsta | S(5) |
| | | Dorman | S(3) |
| | | Dunfield | S(3) |
| Leguminosae (continued) | | Earlyana | S(4) |
| | | Ebony | S(2) |
| | | F.C. 33243 | R(1.3) |
| | | Gatan | S(4) |
| | | Georgian | S(4) |
| | | Habaro | R(1.8) |
| | | Haberlandt | R(1.8) |
| | | Hakote | S(5) |
| | | Harosoy | S(3) |
| | | Hawkeye | S(2) |
| | | Hill | S(2) |
| | | Hood | S(3) |
| | | Illini | S(2) |
| | | Improved Pelican | S(4) |
| | | Jackson | R(1.4) |
| | | Jogun | S(3) |
| | | Kabott | S(4) |
| | | Laredo | R(1.2) |
| | | Lee | S(3) |
| | | Lincoln | S(3) |
| | | Mamloxi | S(5) |
| | | Mammoth Yellow | S(4) |
| | | Mandarin | R(1.9) |
| | | Mendota | R(1.6) |
| | | Midwest | S(4) |
| | | Monroe | R(1.5) |
| | | Morse | S(4) |
| | | Mukden | R(1.5) |
| | | Norredo | S(4) |
| | | Ogden | S(2) |
| | | Otootan | S(4) |
| | | Palmetto | S(2) |
| | | Patoka | S(3) |
| | | Peking | R(1.5) |
| | | Perry | S(4) |
| | | Richland | S(3) |
| | | Roanoke | S(2) |
| | | Sioux | S(3) |
| | | Tanner | S(4) |
| | | Virginia | S(5) |
| | | Wabash | S(3) |
| | | Wilson | S(4) |
| | | Wood's Yellow | S(4) |
| | | Yelnando | S(4) |
| Lotus corniculatus | bird's-foot trefoil | Empire | S(3) |
| | | Mansfield | S(3) |
| Solanaceae | | | |
| Lycopersicon esculentum | tomato | Garden state | S(5) |
| | | Rutgers | S(5) |
| | | Southland | S(5) |
| Curcurbitaceae | | | |
| Cucumis melo | cantaloupe | Delaware Queen | S(5) |
| | | Hale's Jumbo | S(5) |
| | | Hearts of Gold | S(5) |
| | | Schoon's Hard-shell | S(5) |

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# INFLUENCE OF SOME ENVIRONMENTAL FACTORS AND GROWTH SUBSTANCES ON THE DEVELOPMENT OF BARLEY YELLOW DWARF

G. B. Orlob and D. C. Arny

## Summary

Symptoms of barley yellow dwarf were more pronounced under cool temperatures, long days, and high light intensity. These tendencies existed in both susceptible and resistant oat and barley varieties, but resistant varieties were always less affected than susceptible ones. Stunting of susceptible barley plants could be partially overcome by applying gibberellic acid, but treated plants did not reach the size of treated, healthy plants indicating that the "stunting principle" still operated. Infected barley plants sprayed with 3-indoleacetic acid revealed characters indicative of an increased tolerance.

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Limited literature on the influence of temperature and light on the development of small-grain plants infected with barley yellow dwarf virus (BYDV) is available. Wilson and Murphy (10) were first to show that a definite temperature-sunlight interaction determined symptom expression of BYDV in oats, the disease being more severe with low temperatures and direct sunlight. Endo (3) found that shading of infected oats, barley, and wheat plants lengthened the incubation period and decreased symptom severity and that symptoms were severe at 19° and 24°C and progressively less severe at 28° and 31°.

Whereas the influence of temperature and light upon the disease development of virus-infected plants is well established, less is known about other factors which may influence the virus-host interaction. Many growth regulating substances influence profoundly the biochemical and physiological processes in higher plants. Their capacity to induce changes in disease susceptibility has been shown in several instances, primarily in cases of fungal diseases. However, in tobacco mosaic virus, Helms and Pound (4) found that virus concentration, symptom severity, and plant growth decreased when 3-indoleacetic acid (IAA) was added to the nutrient solution in which plants were growing. Black and Lee (2) showed that IAA stimulated symptom development in yellow sweetclover infected with wound tumor virus. Bawden (1) stated that applying auxins usually enhanced symptoms rather than counteracting them. Maramorosch (6) found that stunting in plants infected with aster yellows and other viruses could be overcome by three applications of gibberellic acid (GA), but that diseased plants retained all other signs of virus infection. Similar results have been reported in work with tobacco etch virus (1).

The purpose of the present study was to follow symptom expression of different oat and barley varieties as affected by temperature and light conditions, and by application of certain growth substances.

## MATERIALS AND METHODS

Various oat and barley varieties were grown at 20°C until inoculated with BYDV in the 2 to 3 leaf stage. After an inoculation feeding of 1 to 2 days, viruliferous aphids of Rhopalosiphum fitchii (Sand.) - R. padi (L.) complex and Macrosiphum dirhodum (Wlk.) were removed by a 0.1% malathion spray[1]. Inoculated and non-inoculated checks then were placed in the environment being investigated. For photoperiodic studies, wooden frames covered with black cloth were used for appropriate periods of time. Light intensity was studied in a fluorescent light chamber, various intensities being obtained by covering groups of plants with different layers of cheesecloth on a wooden frame. Light intensities were estimated by means of a light meter such as is used in photography. After 4 weeks under experimental conditions, the plants were harvested and the following measurements taken: 1) average height of shoots, 2) average fresh weight of shoots, and 3) average fresh weight of roots (3). From these data an index was figured:

$$\frac{1 + 2 + 3 \text{ of the infected set}}{1 + 2 + 3 \text{ of the control}} \times 100$$

[1] malathion = S-(1,2-bis(ethoxycarbonyl)ethyl)O,O-dimethyl phosphorodithioate.

Growth substances were dissolved in distilled water, with a few drops of Tween detergent added. The solutions were applied by means of a sprayer to plants at various times after inoculation. The control plants were sprayed with distilled water. C.I. 666 served as a test plant.

## RESULTS

Temperature and Light Intensity: Four oat varieties were used, Coast Black and Clintland as susceptible varieties, and Saia and C. I. 6668 as more resistant varieties. As shown in Table 1, symptom severity generally decreased with rising temperatures, in tolerant as well as susceptible varieties.

Table 1. Influence of four temperatures and four light intensities on the development of barley yellow dwarf in oats.

| Environment | Disease index[a] for given oat variety | | | |
|---|---|---|---|---|
| | Coast Black | Clintland | Saia | C.I. 6668 |
| A. Temperature (°C) | | | | |
| 16 | 41 | 42 | 75 | 63 |
| 20 | 32 | 40 | 78 | 71 |
| 24 | 34 | 41 | 79 | 82 |
| 28 | 50 | 61 | 84 | 85 |
| B. Light Intensity (ft-c) | | | | |
| 5600 | 27 | 48 | 77 | 72 |
| 2200 | 33 | 56 | 80 | 77 |
| 1800 | 45 | 58 | 85 | 78 |
| 600 | 72 | 75 | 87 | 87 |

[a] Index is based on the mean of four experiments.

$$\text{Index} = \frac{(\text{height} + \text{wgt. shoots} + \text{wgt. roots}) \text{ infected plants}}{(\text{height} + \text{wgt. shoots} + \text{wgt. roots}) \text{ non-infected}} \times 100$$

Light intensity seemed to have a more pronounced influence than either temperature or photoperiod. This was especially expressed in the susceptible varieties (Table 1). Symptom expression was most severe at the high intensity, decreasing with diminishing light intensities. As in the case of temperature, the resistant varieties were affected to a lesser extent by differences in light intensities.

Photoperiod: A long day gave more severe symptoms in the four barley varieties, although even under a relatively short day of 8 hours, symptoms were still quite distinct (Table 2).

Table 2. Influence of photoperiod on the development of barley yellow dwarf in barley at 20° C.

| Day length (hours) | Disease index[a] of given barley variety | | | |
|---|---|---|---|---|
| | C.I. 666 | Wis. Ped. 38 | Kindred | C.I. 2376 |
| 8 | 30 | 44 | 69 | 76 |
| 15 | 24 | 29 | 65 | 70 |

[a] See Table 1, footnote a.

Gibberellic Acid: Gibberellic acid was used in concentrations of 10 to 100 ppm. In the first experimental series it was applied five times (at 3- to 4-day intervals) to barley infected with BYDV. Infected plants treated with GA resumed growth and reached almost the size of the non-sprayed, non-infected control plants (Fig. 1). However, non-infected checks sprayed with GA were much taller, indicating that the "stunting principle" still operated in treated, infected plants. In diseased plants a GA concentration of 10 ppm was more effective than 100 ppm. All treated plants, healthy or inoculated, became spindly, and root development was inhibited (Fig. 1). No change in symptom expression other than the reversal of stunting could be observed.

A second experimental series was designed to test the influence of a preinfection treatment on disease development. GA (10 ppm) was applied twice at intervals of 4 days before plants were inoculated, and three additional applications were made after inoculation. Approximately the same results as in the previous experiment were obtained. Infected, sprayed plants were still much shorter than the healthy, sprayed checks.

Indoleacetic Acid: 3-indoleacetic acid (IAA) was used in concentrations of 1 and 10 ppm and sprayed on plants four times at weekly intervals beginning 25 days after inoculation. The 10 ppm solution was found to give maximum response (Fig. 2). Besides a reduction in stunting, treated barley showed less leaf yellowing and a greater leaf expansion, all indicative of increased tolerance.

## DISCUSSION

FIGURE 1. Influence of five postinfection sprays of gibberellic acid at a concentration of 10 ppm on the development of healthy and BYDV-infected barley. From left to right: healthy, sprayed; healthy, non-sprayed; infected, sprayed; infected, non-sprayed.

Changes in environmental conditions have been shown to affect susceptibility and symptom expression of virus-infected plants, and the extent to which viruses increase in the host. Each virus-host combination may react differently to a given environment; certain virus diseases develop symptoms over a wide range of temperatures, whereas symptom expression of other diseases is limited to a narrower range of temperatures, being masked otherwise. BYDV clearly belongs to the former group of virus diseases, since symptoms could be recognized over a wide range of environmental conditions.

Furthermore, BYDV shows characteristics of the group of aphid-borne viruses producing yellowing or leaf rolling symptoms. It is generally accepted that low light intensities reduce symptoms of plants infected with viruses of this class (5). Kassanis (5) believed that rolling of leaves of leaf-roll-infected potatoes or yellowing of yellows-infected beets was entirely a function of light intensity. Watson (8) demonstrated that the severity of symptoms caused by sugar beet yellows could be decreased by reducing light intensity. In potato leaf roll the same tendency was observed by Webb, et al. (9). In the case of BYDV, the work of Endo (3), Wilson and Murphy (10) and the present study were in a similar direction.

At present little work has been done on plant virological aspects of the gibberellins, and nothing is known about the underlying mechanism of the reversal of virus-induced stunting. A comparable case of stem elongation is found in the effect of GA on genetic dwarfs (7), and the mechanism of growth stimulation could be quite similar to that in virus-induced dwarfs. Also, it was found that not all genetic dwarfs responded to a GA treatment, just as not all virus-stunted plants react alike.

From the data available, it seems that other growth substances, such as IAA, influence a given virus plant combination differently, either depressing symptoms and virus multiplication or exerting synergistic effects. The different reaction of hormone-treated, virus-infected plants could be explained through a specific effect of the growth substance on the metabolism of the host, and through it, on virus infection, multiplication, or movement.

FIGURE 2. Influence of four applications of indoleacetic acid on the development of healthy and BYDV-infected barley. Left to right: infected plants sprayed with IAA at a concentration of 10 ppm; infected plants sprayed at a concentration of 1 ppm; non-sprayed infected plants; non-infected plants sprayed with IAA at a concentration of 10 ppm; non-sprayed healthy plants.

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# HYPOXYLON TINCTOR ASSOCIATED WITH A CANKER ON AMERICAN SYCAMORE TREES IN GEORGIA

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American sycamore (Platanus occidentalis) is an important bottom-land species in the Georgia Piedmont. The wood is used in the manufacture of a variety of products but finds its greatest market in the furniture industry. The tree grows generally in admixture with other bottom-land species but often is also found in almost pure groups usually not exceeding an acre or two in size. Thinning or improvement cutting is often desirable in the rather dense even-aged groups.

In 1955 an improvement cut was made in a 30-year-old stand, 3 acres in size, located in the overflow bottom lands of the Oconee River in Greene County, Georgia. Sample trees within 1/5-acre plots were carefully examined annually for defects, usually epicormic branches, which might result from opening the canopy.

In 1958 several trees were found with cankers similar in external appearance to canker stain caused by Endoconidiophora sp. (now included in Ceratocystis fimbriata Ell. & Halst.) (3). In each case, however, there was profuse fruiting of Hypoxylon tinctor (Berk.) Cke. on the canker (Fig. 1)[2]. Toole[3] reported a stem canker on sycamore in the Mississippi Delta, but H. tinctor was not associated with it.

In 1960, 20 trees within and immediately surrounding the cut areas had cankers, and in each case H. tinctor was associated. Eight of these trees were dead and several others were dying. The average diameter of the affected trees was 13 inches at breast height, with trees as small as 6 inches and as large as 30 inches in diameter being affected. Cankers occurred on trees irrespective of size, vigor, or crown class.

Most cankers occurred at some distance above ground on the main stems, implying the possibility of infection at branch stubs. General weakening of the wood in the area of the canker (Fig. 2) resulted in eventual loss of the top portions of the trees. Half of the infected trees had broken over at heights of from 15 to 50 feet above the ground level, and several had cankers extending from the ground to as high as 40 feet and encompassing over one-third of the circumference of the bole. Cankers generally followed the grain of the wood, in some cases twisting almost completely around the bole.

Hypoxylon tinctor has been reported many times as a saprophyte on sycamore and other hosts (1). This is apparently the first time the organism has been found associated with a canker on living trees. Examinations of other hosts--green ash, boxelder, hackberry, and hickory in admixture with the sycamore--did not disclose any fruiting of H. tinctor on either living or dead trees. However, other Hypoxylon species were fruiting on logs of bitternut hickory (Carya cordiformis) left from the 1955 cutting.

In 1951 Thompson (2) reported a dieback of sycamore near Athens, Georgia, caused by Diplodia natalensis Evans. This disease affected the small branches and spread into the larger limbs and bole, often killing the tops of trees. The canker associated with H. tinctor appears to affect the main stem directly, and it may cause the top to die and eventually break over. The advancing margin of the canker is irregular and rather indistinct. The bark in the area of the canker is light orange-brown and may be slightly sunken in the older cankers. Often longitudinal cracks appear at points of stress.

These observations show only that Hypoxylon tinctor is associated with a canker causing mortality of sycamore trees. No studies have been made to determine pathogenicity. This unforeseen development following rather heavy thinning in a sycamore stand may prove to be an important factor in future culture of sycamore in the Georgia Piedmont.

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[2]All Hypoxylon species were determined by Dr. Julian H. Miller.

[3]Unpublished report, U. S. Forest Service, Southern Forest Experiment Station, New Orleans.

FIGURE 1. Hypoxylon tinctor fruiting on a 12-inch living sycamore tree.

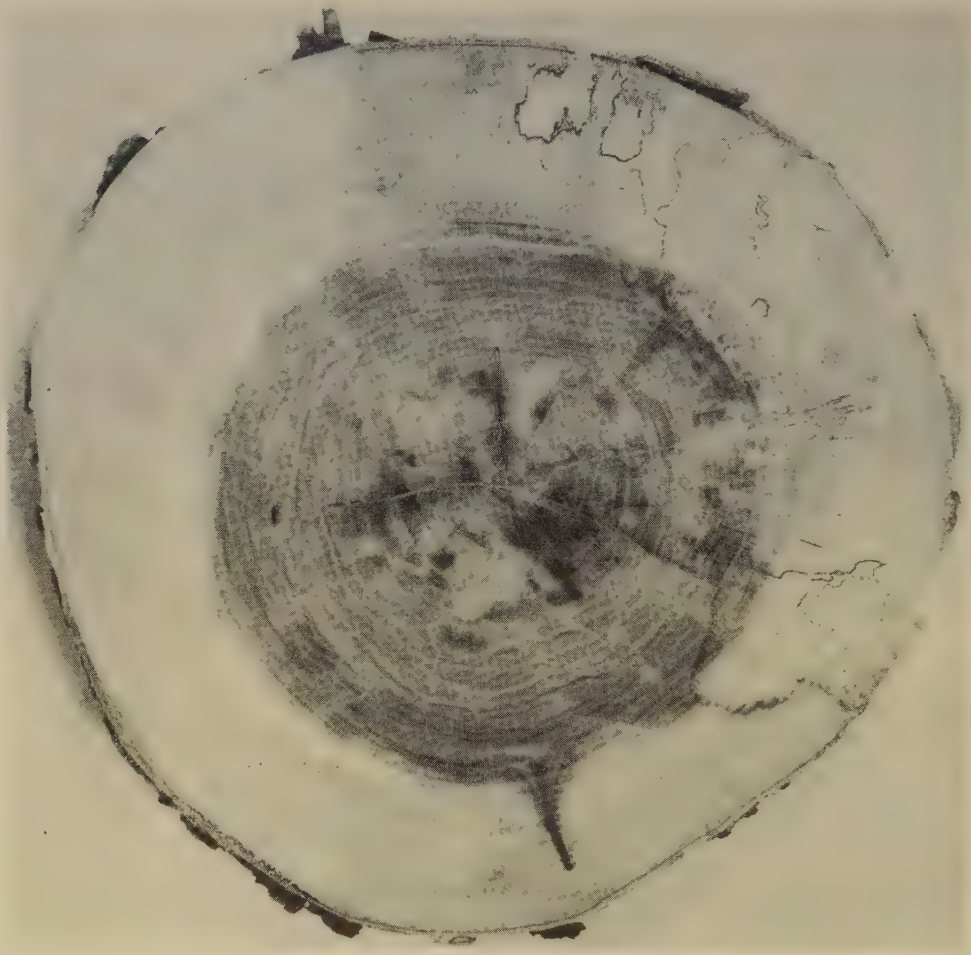

FIGURE 2. Cross section of a 10-inch living sycamore showing irregular and rather indistinct advancing margin of canker.

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# THREE ADDITIONAL BROMEGRASS SPECIES AS HOSTS OF HELMINTHOSPORIUM SOROKINIANUM (H. SATIVUM)

Samuel W. Braverman[1]

Abstract

Helminthosporium sorokinianum Sacc. ex Sorokin (H. sativum Pam., King, & Bakke) was isolated from several Bromus species in a seed-plot nursery at the Regional Plant Introduction Station, Geneva, New York in 1960. The causal fungus is described on three previously unreported bromegrass species, Bromus brevis, B. tomentosus, and B. frondosus.

Helminthosporium sorokinianum Sacc. ex Sorokin (H. sativum Pam., King, & Bakke) was repeatedly isolated from numerous bromegrass species in a seed-plot nursery at the Regional Plant Introduction Station in 1960. The fungus previously was reported pathogenic on 20 Bromus spp. by greenhouse and field inoculations[2] and on naturally occurring hosts[3]. However, B. brevis (P.I. 202014), B. tomentosus (P.I. 229603), and B. frondosus have not been included.

Leaves of the three species were placed in Petri dish moist chambers for 48 hours. Conidia of the H. sorokinianum type were found in abundance on the diseased leaf areas. The fungus also sporulates profusely on potato-dextrose agar slants. Generally, conidia are dark olive-brown, taper slightly at both ends, and occasionally are curved. The hilum is somewhat conspicuous and is enclosed within the exospore. Conidial measurements are presented in Table 1.

Table 1. Measurements of conidia of H. sorokinianum isolated from leaves of three bromegrass species.

| Host | Size of conidia (in $\mu$) | | Septa | |
|---|---|---|---|---|
| | Range | Average | range | Average |
| B. brevis | (65-97) x (19-24) | 79 x 22 | 4-9 | 6.8 |
| B. tomentosus | (62-86) x (19-24) | 77 x 22 | 5-9 | 7.1 |
| B. frondosus | (62-78) x (17-24) | 68 x 21 | 4-8 | 6.1 |

Symptoms on leaves consist of irregular, dark brown spots (3 x 22 mm long) or blotches with a definite margin. Immature lesions are surrounded by a faint yellow margin. Frequently symptoms may coalesce, leaving extensive areas of necrotic leaf lamina.

This constitutes the first report of Helminthosporium sorokinianum on B. brevis, B. tomentosus, and B. frondosus.

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# A NEW BROAD SPECTRUM SOIL FUNGICIDE[1]

Wayne R. Sitterly[2]

The intensive culture of vegetable crops in the coastal area of South Carolina makes control of several soil fungi an economically important factor in successful crop production. The planting of many crops in cool, wet soil in winter or early spring requires a good seed protectant. Cucumber yields have been reduced as much as 30% by fruit rot caused by Pythium and Rhizoctonia species. Frequently tomato yields are reduced by stem and fruit rots caused by Sclerotium rolfsii and Rhizoctonia solani. Bean yields are often decreased by Rhizoctonia root rot and Pythium damping-off. These are but a few examples of the problems which have made the search for effective soil fungicides a necessary part of any pesticide program in this area. This report is concerned with the results obtained from the use of the soil fungicide 5-chloro-4-phenyl-1,2-dithiol-3-one (H3944)[3] developed by the Hercules Powder Company of Wilmington, Delaware.

## MATERIALS AND METHODS

Seed Treatment: Seeds of beet, lettuce, carrot, spinach, onion, pea, okra, watermelon, cucumber, lima bean, and tomato were treated with nine different chemicals at recommended rates and stored at 70° F for 30 days before planting. Each crop was planted at the season of the year when it is normally planted in the Charleston area. Data were recorded 10 days after seedling emergence.

After preliminary tests had indicated the effectiveness of H3944 as a seed protectant against various soil-borne decay organisms, it was decided to test this material against other important soil-borne pathogens, namely, Sclerotium rolfsii on tomatoes and Pythium and Rhizoctonia spp. on cucumbers.

Tomato Soil Fungicides: In an effort to control Sclerotium rolfsii, five chemical treatments were applied to tomatoes at different stages of plant development. These materials were applied either in the transplant water or onto the soil and thoroughly mixed with the soil at designated periods.

Cucumber Soil Fungicides: Ten treatments were applied to the soil around cucumber plants immediately after the last cultivation in an attempt to reduce fruit rot losses caused by Pythium and Rhizoctonia. Chemicals were applied in 200 gallons of water per acre at 200 pounds/square inch pressure. An area 2 feet wide on either side of the plants was covered by means of a directed spray. The treated area was left undisturbed throughout the harvest season. Normal rainfall during the harvest season was supplemented with irrigation in order to provide the wet conditions most favorable for fruit rot development.

General Procedures: Randomized block designs with from five to six replicates were used in all tests. Tomato and cucumber trials were conducted both in the spring and fall seasons in 1960. All soil fungicide plots received recommended cultural operations, including routine applications of foliage fungicides and insecticides. Cucumber and tomato yield and plant loss data were recorded at the normal harvest dates.

## RESULTS

Seed Treatment: H3944 appeared to be at least as effective as any of the chemicals used as a seed protectant on the 11 vegetable crops (Table 1). Seven of the 11 crops showed a significant increase in stand because of seed treatment by one or more of the chemicals. All seven responded significantly to H3944. Six of the seven responded to treatment with tetramethylthiuram disulfide (thiram) and five to treatment with pyridinethione 1 oxide, zinc salt (Omadine zinc). N-trichloromethylmercapto-4-cyclohexene-1,2-dicarboximide (captan) and tetrachloro-para-benzoquinone (chloranil) were slightly less effective. The other four chemicals tested were even less effective and the results from these were omitted from Table 1.

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[1]Technical Contribution No. 357, South Carolina Agricultural Experiment Station, Clemson College, Clemson, S. C. Published by permission of the Director.

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[3]H3944 has been produced for experimental purposes only and is not available in commercial quantities.

Table 1. The effect of various seed protectants on germination of winter and early spring seeded vegetable crops at Charleston, South Carolina, in 1960.

| Crop | % germination[a] | | | | | |
|---|---|---|---|---|---|---|
| | Untreated | Captan | Thiram | Chloranil | Omadine zinc | H3944 |
| Beet | 16 | 15 | 32 | 11 | 26 | 32 |
| Lettuce | 21 | 62 | 70 | 68 | 65 | 54 |
| Spinach | 20 | 54 | 61 | 43 | 64 | 66 |
| Onion | 60 | 68 | 67 | 60 | 65 | 73 |
| Carrot | 16 | 35 | 51 | 23 | 36 | 42 |
| Pea | 94 | 97 | 96 | 97 | 97 | 93 |
| Watermelon | 53 | 63 | 70 | 62 | 72 | 78 |
| Cucumber | 57 | 66 | 64 | 60 | 57 | 70 |
| Okra | 37 | 59 | 62 | 56 | 62 | 62 |
| Lima bean | 9 | 31 | 52 | 27 | 56 | 50 |
| Tomato | 59 | 53 | 63 | 65 | 55 | 60 |

[a] Underlined figures represent a significantly better stand than was obtained from the use of untreated seed.

Table 2. The effect of certain chemicals applied at various times of plant development on the control of southern stem blight of tomato caused by Sclerotium rolfsii.

| Chemical | Rate (pounds/acre) | Method of application | Time of application | % infected plants | |
|---|---|---|---|---|---|
| | | | | spring | fall |
| Control | | | | 65 | 30 |
| PCNB (Terraclor) 40% | 18 | Broadcast | Preplant | 47 | 5 |
| PCNB (Terraclor) 75% | 4 | Transplant water | At transplant | 28 | |
| $Ca(NO_3)_2$ | 100 | Around plant base | At transplant | 22 | 7 |
| Hercules 3944 | 4 | Transplant water | At transplant | 17 | 13 |
| $Ca(NO_3)_2$ | 100 | Side-dress in alley | Last cultivation | 35 | |
| $Ca(NO_3)_2$ | 100 | Around plant base | Last cultivation | 40 | 23 |
| Basic slag | 200 | Around plant base | Last cultivation | 45 | |
| $CaSO_4$ | 200 | Around plant base | Last cultivation | 65 | |
| Least Significant Difference at 5% point | | | | 11 | 9 |
| Least Significant Difference at 1% point | | | | 17 | - |

Tomato Soil Fungicides: When H3944 was applied in tomato transplant water in the spring of 1960, only 17 % of the treated plants became infected with Sclerotium rolfsii as compared with 65% in the untreated plots (Table 2). This difference was statistically highly significant. Only $Ca(NO_3)_2$, applied as a side-dressing at transplanting, appeared to be as effective, and there have been conflicting reports concerning this material (1,3). Similar results were obtained from a fall crop, except that pentachloronitrobenzene (PCNB) (Terraclor) applied broadcast to the soil prior to setting the plants gave equally good control to that obtained with H3944 and $Ca(NO_3)_2$.

Although the originally treated area was repeatedly covered with untreated soil until a sufficiently high plant bed was obtained, H3944 was still effective. Pelletier and Palmer (2) demonstrated that, although H3944 is not volatile, it does become highly active biologically upon being mixed or covered with soil. Apparently this biological activity was retained for an extended period, which explains the degree of control of Sclerotium rolfsii obtained.

Cucumber Soil Fungicides: H3944 was the only treatment that effected a significant reduction in the amount of cucumber fruit decay below the untreated control both in the spring and in the fall crops (Table 3). In the fall, H3944 at 8 pounds/acre proved at least as effective or slightly more effective than mixtures of H3944 with either captan or N-trichloromethyl thiophthalimide (Phaltan), each at 4 pounds/acre. The number of diseased fruits was very high, especially in the spring, because of the ideal conditions for decay provided by the supplemental irrigation. H3944 and a paper mulch treatment gave significant yield increases in the spring crop and all treatments gave significantly increased yields in the fall. H3944 produced some

Table 3. The effect of certain treatments on cucumber yields and on the control of Pythium and Rhizoctonia fruit rot.

| Treatment[a] | Rate (pounds/acre) | Spring trial yield (bushels/acre) | Spring trial fruit rot (%) | Fall trial yield (bushels/acre) | Fall trial fruit rot (%) |
|---|---|---|---|---|---|
| Control | | 172 | 48.2 | 145 | 21.8 |
| Captan 50% | 8 | 195 | 48.6 | 216 | 19.1 |
| PCNB 75% | 8 | 158 | 46.4 | | |
| Hercules 3944 50% | 8 | 200 | 40.0 | 212 | 10.8 |
| Phaltan 50% | 8 | 175 | 46.0 | 221 | 14.8 |
| Captan + Phaltan | 4+4 | 158 | 48.2 | | |
| Captan + PCNB | 4+4 | 112 | 56.0 | | |
| Captan + H3944 | 4+4 | | | 236 | 13.8 |
| Phaltan + PCNB | 4+4 | 164 | 45.2 | 214 | 15.8 |
| Phaltan + H3944 | 4+4 | | | 234 | 13.2 |
| Westvaco paper | | 208 | 43.2 | 227 | 16.4 |
| Black plastic | | 173 | 44.4 | 212 | 15.8 |
| Least Significant Difference at 5% point | | 27 | 8.0 | 51 | 4.8 |
| Least Significant Difference at 1% point | | 38 | 10.9 | - | 6.9 |

[a] All treatments applied to a strip 24 inches wide on either side of the row immediately following the last cultivation.

marginal leaf scorching of the older leaves, but the plants recovered apparently without permanent injury. This is in contrast to the permanent damage to the foliage caused by applications of PCNB.

## CONCLUSIONS

The fungicide H3944 appeared to be effective against several soil fungi. Effectiveness did not seem to be limited because of the method of application: H3944 was effective against seed decay organisms when applied as a seed treatment in dust form; it was effective against Sclerotium rolfsii when applied to tomatoes in the transplant solution; and it was effective against Rhizoctonia and Pythium species when applied as a spray on the soil surface around cucumbers after the last cultivation.

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# LEAF BLIGHT OF DRACAENA INCITED BY PHYLLOSTICTA DRACONIS

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During the month of August 1958, black specks, mostly in concentric layers, were observed on both sides of bleached Dracaena leaf at the Institute of Agriculture, Anand. Infection was chiefly confined to older leaves while young leaves at the top were free from infection. The affected area first becomes yellow, irregular in contour and later turns brown with dark violet margin (Fig. 1b). Pycnidia develop in discolored area of the leaf (Fig. 1a).

FIGURE 1. Symptoms caused by Phyllosticta draconis Berk. on Dracaena sp.

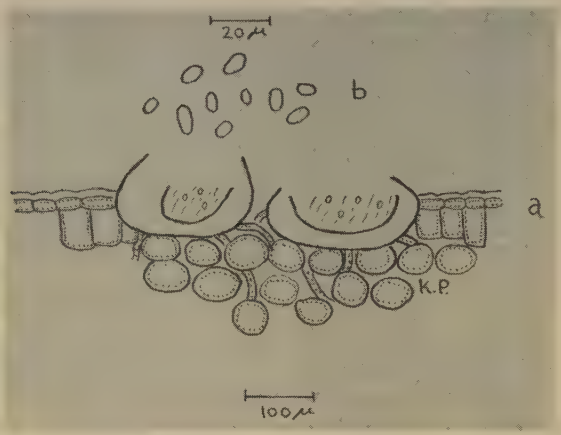


FIGURE 2. a) Locating position of pycnidia of Phyllosticta draconis Berk. on Dracaena sp. b) Spores of Phyllosticta draconis Berk.

Microscopic study of the infection spot revealed numerous, subepidermal and globose to lenticular pycnidia which were erumpent (Fig. 2a). The pycnidia were thinwalled, ostiolate, measuring from 63-94.5μ x 112.5-132.3μ. Spores were small, measuring 2.2-3.08μ x 5.94-10.12μ, oblong and hyaline (Fig. 2b). Conidiophores were indistinct.

Diseased specimens of Dracaena collected from Bombay, Poona, Banglore and Mysore showed the same fungus, thereby indicating its wide distribution.

Saccardo (4) has recorded leaf spots on Dracaena caused by Phyllosticta draconis Berk. The fungus has been shown to be a serious pathogen in Portugal on Dracaena draco and in Great Britain on D. terminalis and D. cooperi (3). Griffon and Maublanc (2) described the fungus causing leaf spots on cultivated Dracaena and named it Phyllosticta dracaenae. Evidently daCosta and Mundkur (1) reported P. dracaenae on Dracaena from India on the basis of descriptions given by Griffon and Maublanc. The descriptions of the organisms described by Saccardo and by Griffon and Maublanc are identical. Griffon and Maublanc said, "this was not still known. We describe it under the name of Phyllosticta dracaenae." Hence there remains no doubt that Griffon and Maublanc had missed the earlier description by Saccardo.

In the present case the disease symptoms appear as a leaf blight somewhat different from those that have already been described by Saccardo, and by Griffon and Maublanc. The disease symptoms indicate the nature of host reaction. Since the fungus described by Saccardo and Griffon and Maublanc are indistinguishable morphologically, P. dracaenae Griff. & Maubl. may prove to be synonymous with P. draconis Berk.

The authors are grateful to Dr. M. J. Thirumalchar for helpful suggestions.

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# THE HOST RANGE OF THE VIRUS OF LAMBERT MOTTLE OF CHERRY, A PROGRESS REPORT[1]

T. B. Lott and F. W. L. Keane[2]

## Abstract

Five selections of the virus of Lambert mottle, obtained from naturally infected trees, were inoculated into various stone and pome fruits in 1952 and 1953. Fourteen varieties of sweet cherry (Prunus avium on P. avium) became definitely infected, and 11 varieties expressed symptoms similar to those in Lambert. On P. mahaleb rootstocks, three varieties produced results similar to those on P. avium. Infection, but without recognizable symptoms, was demonstrated in sour cherry (P. cerasus on P. mahaleb), peach (P. persica), and rootstock seedlings (P. mahaleb). No indications of subsequent infections were obtained when the virus of Lambert mottle was inoculated into chokecherry (P. virginiana var. demissa), apricot (P. armeniaca on P. armeniaca), plum (P. domestica on P. cerasifera), wild cherry (P. emarginata var. mollis), four varieties of apple and one variety of pear.

## INTRODUCTION

Lambert mottle (1, 2) appeared in 1939 in four young Lambert sweet cherry trees which had been inoculated with mottle leaf (3) obtained originally from a Napoleon (Royal Ann) tree in the Kootenay area of British Columbia. The same year Lambert mottle was found in two commercial Lambert trees in the Okanagan Valley. Between 1938 and 1941, orchard surveys covered more than 9000 cherry trees in the Okanagan Valley but revealed only nine Lambert trees naturally infected with Lambert mottle. Transmission gave symptoms in mature leaves in 1943 and early season symptoms in 1944. The disease was then recognized as being distinct from mottle leaf. It appeared to be of little importance until 1945 and 1946 when orchards in Westbank and Kelowna were found to contain a disease which was similar in appearance but quicker acting and more damaging. This disease was reported to have ruined almost all the Lambert trees in some orchards in about 10 years. Other varieties seemed to be unaffected. More recently other Lambert trees have been found in various parts of the valley with symptoms similar but sometimes not identical.

Inoculations were made in 1952 and 1953 into a range of possible hosts, and the results are presented here. No attempt is made to relate the disease or diseases in British Columbia to any that occur elsewhere.

## MATERIALS AND METHODS

Five selections of Lambert mottle were obtained from commercial cherry trees and were inoculated into fifteen varieties of sweet cherry (Prunus avium on P. avium), into three of the same varieties on P. mahaleb, into sour cherry (P. cerasus on P. mahaleb), into peach seedlings (P. persica), into rootstock seedlings (P. mahaleb), into trees of the native chokecherry (P. virginiana var. demissa), into apricot (P. armeniaca on P. armeniaca), into plum (P. domestica on P. cerasifera), into trees of the native wild cherry (P. emarginata var. mollis), into four varieties of apple and one variety of pear.

The five selections of Lambert mottle were obtained from Lambert trees in different parts of the Okanagan Valley. Selection 1, obtained in Summerland, came from the orchard where naturally occurring Lambert mottle was first observed, and it was typical of the disease as described in 1945 (1). Selection 2 came from East Kelowna where very serious damage had occurred in some orchards. It showed some of the leaf pattern found in Selection 1, but produced leaf symptoms in May instead of June, had larger continuous burned patches in the leaf blades with defoliation, and caused the death of large branches within a year. In some large trees the roots and trunk remained alive while almost the entire top was killed in 2 years. Selection 3 came from an East Kelowna orchard and was similar to Selection 2 but with much less leaf burning. Defoliation was mainly confined to the lower leaves. Strong terminal growth

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was followed by very extensive branch killing. Selection 4 came from Osoyoos. The orchard tree had rather small and yellowish-green leaves. They were nearly covered with papery spots up to 3/8 inch, and most of them were distinctly curled. Defoliation became almost complete in the summer and was followed by very extensive killing of the branches. This tree did not show the leaf pattern associated with slow-acting Lambert mottle which had been present to a small extent in this orchard for about 10 years. Selection 5, from Westbank, showed symptoms intermediate between those of Selections 1 and 4.

All five selections of diseased material and most of the sweet cherry test trees gave positive reactions on Shiro-fugen.

Inoculations, direct from the commercial trees, were made with Selections 1 and 2 in 1952, and with Selections 3 and 5 in 1953. Inoculations with Selection 4 were made in 1952 with inoculum from the surviving mazzard rootstock in an experimental tree in which the Lambert part had been killed by the disease.

The plot had been originally designed for the inoculation of each selection into paired trees of each kind. However, the experiment was twice affected by serious winter damage. Many plot trees were lost in the severe winter of 1949-50 and this damage was only partially overcome, so that some trees were still missing at the time of inoculation. Weather and growth conditions then remained favorable until the most important observations had been completed in the summer of 1955. A severe frost occurred in November 1955 and was followed by others during the winter. With the exception of apple and pear, each experimental tree was inoculated with a single selection of Lambert mottle. Inoculations were made by inserting two buds from the inoculum tree into the plot tree.

Ten selections of Lambert mottle, including Selections 1 and 4, were inoculated together in 1954 into four apple trees, of the Delicious, Jubilee, Spartan and Winesap varieties, and into seven Bartlett pears. Backcheck inoculations were made annually from 1955 to 1958 out of these apples and pears individually into young Lambert trees.

Backcheck inoculations into Lambert were made out of all plot trees except Lamberts, and except trees inoculated with Selection 5. Backcheck inoculations were made satisfactorily out of inoculated sour cherry, peach, mahaleb, chokecherry, apricot, plum, and wild cherry trees, in 1955. Backcheck inoculations out of sweet cherry trees were much less satisfactory because they were made in the spring and summer of 1956 out of trees which were in many cases seriously winter injured.

## RESULTS

Attempts were made to determine both the ability of the different kinds and varieties of trees to become infected with the virus of Lambert mottle, and their ability to display symptoms of the disease. Lambert trees on both mazzard and mahaleb rootstocks produced satisfactory symptoms. Diagnosis in other varieties of sweet cherry was more difficult because no variety other than Lambert was found to produce symptoms which could be recognized with certainty. Some varieties did produce leaf and twig symptoms similar to those in Lambert though not identical. Some young experimental trees died apparently but not certainly as a result of infection. The situation was further confused by the winter injury that started in 1949.

The definite determination of the presence of Lambert mottle virus depended on the observation of symptoms in the Lambert variety. Hence, infection with Lambert mottle virus was considered certain only in cases where recognizable disease was produced by inoculation into a Lambert backcheck tree. However, it was considered that all the inoculated sweet cherry trees in the plots had, each tree individually, a high probability of being infected, even symptomless trees which did not give positive backchecks. This assumption seems to be justified by the definite infection of all the 12 Lambert trees shown in Table 1, and by the considerable number of positive backchecks obtained out of sour cherry, peach, and mahaleb, as well as out of symptomless sweet cherry.

The results of inoculating the five selections of Lambert mottle into 15 varieties of sweet cherry are presented in Table 1. All other species remained symptomless after inoculation. Of these, sour cherry, peach, and mahaleb were backchecked into Lambert and proved to be hosts of the virus. The remaining kinds, chokecherry, apricot, plum, wild cherry, apple, and pear, were similarly backchecked into Lambert with negative results.

Table 1. The symptom expression of five selections of Lambert mottle virus in varieties of sweet cherry.

| Variety of cherry [a] | Selection 1 | | Selection 2 | | Selection 3 | | Selection 4 | | Selection 5 | |
|---|---|---|---|---|---|---|---|---|---|---|
| | No. of trees | Symptoms | No. of trees | Symptoms | No. of trees | Symptoms | No. of trees | Symptoms | No. of trees | Symptoms |
| Bing | 2 | Symptomless P O [b] | 2 | Symptomless O O [b] | 2 | Symptomless P P [b] | 2 | Symptomless P P [b] | 2 | Symptomless O O [b] |
| Bing on mahaleb | 1 | Symptomless P | 1 | Symptomless P | 2 | Symptomless P O | 2 | Symptomless P P | | |
| Black Republican | 2 | Symptomless P P | 2 | Moderate defoliation P O | | | 1 | Symptomless P | 1 | Symptomless O |
| Black Tartarian | 2 | Symptomless P O | 2 | Symptomless P P | 1 | Symptomless O | 1 | Symptomless P | 2 | Symptomless O O |
| Black Tartarian on mahaleb | 2 | Symptomless O O | 1 | Symptomless P | 1 | Symptomless P | 1 | Symptomless P | 2 | Symptomless O O |
| Deacon | 1 | Slight burn spots and rings O | 2 | Slight pattern and burn P P | | | | | | |
| Hedelfingen | 1 | Symptomless P | | | 1 | Symptomless P | 2 | Symptomless P P | 1 | Symptomless O |
| Napoleon (Royal Ann) | 2 | Symptomless O O | 1 | Symptomless P | 1 | Symptomless P | 1 | Slight pattern O | 1 | Symptomless O |
| Sam | 2 | Slight pattern and burn, severe defoliation P P | 1<br>1 | Symptomless<br>Slight pattern, severe burn and defoliation, killed P P | 1 | Slight pattern, severe burn and defoliation, moderate die-back O | 2 | Severe burn and defoliation and die-back O O | 2 | Slight pattern and burn O O |
| Sparkle | 2 | Slight pattern P O | 2 | Symptomless P O | 2 | Symptomless P P | 2 | Symptomless P P | 2 | Symptomless O O |
| Star | 2 | Symptomless O O | 1 | Slight burn P | | | 1<br>1 | Slight burn severe die-back<br>Slight burn and defoliation, no die-back P O | | |
| Sue | 2 | Slight burn P P | 2 | Slight burn and defoliation P P | | | 2 | Symptomless P P | 2 | Symptomless O O |
| Seedling S-8-4 | 2 | Slight pattern and burn, moderate defoliation P P | 1 | Slight burn and defoliation P | 2 | Symptomless P P | 2 | Slight pattern and burn P P | 2 | Symptomless O O |
| Van | 2 | Symptomless P P | 2 | Symptomless P P | 2 | Symptomless P P | 2 | Moderate burn P P | 1 | Symptomless O |
| Velvet | 2 | Slight pattern and burn and defoliation O O | 1 | Slight pattern and burn and defoliation O | | | 1<br>1 | Killed<br>Severe burn and defoliation, slight die-back P O | 1 | Slight burn O |
| Victor | 2 | Symptomless P O | 1 | Symptomless P | 2 | Symptomless P P | 2 | Slight pattern and burn P P | 2 | Symptomless O O |
| Lambert | 2 | Moderate pattern, slight burn, moderate die-back | 2 | Moderate burn and defoliation, severe die-back | 1 | Slight pattern and defoliation, moderate burn, severe die-back | 2 | Severe burn and die-back, killed | 1<br>1 | Slight burn and die-back<br>Moderate burn, severe die-back |
| Lambert on mahaleb | 2 | Slight pattern and burn, moderate die-back and defoliation | 1 | Moderate pattern, severe burn and defoliation and die-back | | | 1 | Moderate pattern and burn, severe die-back and defoliation, killed | | |

[a] All trees were on mazzard rootstocks unless otherwise stated.

[b] P Virus present as shown by back inoculation into Lambert.
O No satisfactory back inoculation.

## DISCUSSION

This work broadens considerably the known host range of the virus of Lambert mottle. Fourteen of 15 varieties of sweet cherry were proved to have been infected. Symptoms, similar to but not identical with those expressed in Lambert, were produced in 11 varieties. Three of the infected varieties remained symptomless. For three varieties, results were similar on mahaleb and on mazzard rootstocks.

Sour cherry, peach, and mahaleb became definitely infected but without recognizable symptoms of Lambert mottle. The only kinds which gave no indications of susceptibility were chokecherry, apricot, plum, wild cherry, apple, and pear.

The results are not considered to have been seriously affected by Shiro-fugen-positive latent viruses.

This work is being continued using a greater number of selections of Lambert mottle virus on some of the same hosts and a few other species. Incomplete results, already obtained, indicate confirmation and some slight modification of the results reported in this paper.

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# STUDIES ON THE PREVALENCE AND COMPARATIVE PATHOGENICITY OF FUNGI ASSOCIATED WITH CORN STALK ROT[1]

Joseph L. Peterson

## Summary

In 1959 Gibberella zeae (Schw.) Petch, Fusarium moniliforme Sheld., and species of Helminthosporium, Trichoderma, Penicillium, Curvularia, Mucor and Fusarium were isolated from stalk rotted corn in New Jersey. F. moniliforme, G. zeae, H. sativum and Trichoderma sp. were isolated most frequently. Two inbred field corn lines, one susceptible and one resistant, were inoculated with selected isolates of these organisms. Under the conditions tested, F. moniliforme as well as Trichoderma sp., H. sativum, Curvularia sp., and two other species of Fusarium, the latter organisms not commonly thought to be associated with stalk rot, were as pathogenic and in some cases more pathogenic than G. zeae in the susceptible inbred. The resistant inbred was equally resistant to all rot producing organisms tested.

## INTRODUCTION

In the Northeast Region of the United States, Gibberella zeae (Schw.) Petch is generally thought to be the most important cause of stalk rot of corn. Several other organisms are known to cause stalk rot or to be associated in the stalk rot complex in the New Jersey area (1, 3).

This paper presents data on the kind and frequency of fungus organisms isolated from stalk rotted field corn in New Jersey. Results of comparative pathogenicity tests using selected isolates of these organisms are also included.

## PROCEDURE

Isolations were made in 1959 from stalk rotted corn plants selected at random in the field approximately 5 weeks after full silk. Notations were made on the relative prevalence of the various fungal organisms isolated.

In 1960, sixteen isolates were selected from these organisms and compared for pathogenicity in field corn. Two corn inbreds were selected, Os 420, a line of poor standability and considered to be susceptible to stalk rot, and C103, a line of good standability and considered to be fairly resistant to the stalk rot disease. A randomized design was used with 20 plants per replication and three replications of each inbred. Inoculations were made with infested quill type tooth picks. The tooth picks were boiled in water, placed in potato-dextrose broth, autoclaved, and infested with the various selected isolates (4). Inoculations were made by inserting the infested tooth picks into the center of the second fully expanded internode when the corn plant reached full silk. The relative pathogenicity of the isolates was based on the extent of rot in the internode 5 weeks after inoculation. Each internode was split lengthwise and at right angles to the point of insertion of the tooth pick. The linear extent of rot was measured and given a numerical rating according to the percentage of rot. Data obtained from rot readings were averaged within each replication.

## RESULTS

The frequency of isolation of the various organisms follow: F. moniliforme 24%; G. zeae, 20%; H. sativum, 20%; Trichoderma sp., 17%. Species of Fusarium, Mucor, Penicillium and Curvularia, isolated less frequently, accounted for the remaining 19%.

The results of the pathogenicity tests using selected representatives from these organisms are given in Table 1. Three isolates of G. zeae were used in this test and each proved to be quite pathogenic to the susceptible inbred. A species of Trichoderma included in the test produced considerable internode rot in all three replications. The rot caused by this organism was dryer than that usually caused by G. zeae and the rot had a tendency to progress to the outer cortical region of the stalk as well as toward the nodes. Sporulation was often observed in the more rotted areas of the Trichoderma induced rot. Other isolates, particularly in the Fusarium group, caused a rot that followed the vascular strands with less tendency to infect

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Table 1. The relative pathogenicity of stalk rot isolates in susceptible and resistant field corn inbreds.

| Isolate | Replication 1 | | 2 | | 3 | | Average | |
|---|---|---|---|---|---|---|---|---|
| | R[a] | S[a] | R | S | R | S | R | S |
| G. zeae | 0.5[b] | 1.5 | 0.5 | 3.5 | 0.5 | 3.0 | 0.5 | 2.7 |
| G. zeae | 1.0 | 2.0 | 0.5 | 2.5 | 0.5 | 2.5 | 0.7 | 2.3 |
| G. zeae | 1.0 | 2.0 | 0.5 | 2.0 | 0.5 | 2.5 | 0.7 | 2.2 |
| Trichoderma sp. | 0.5 | 3.5 | 0.5 | 3.5 | 0.5 | 3.5 | 0.5 | 3.5 |
| F. moniliforme | 0.5 | 2.0 | 0.5 | 3.0 | 0.5 | 3.0 | 0.5 | 2.7 |
| Fusarium sp. | 0.5 | 2.5 | 0.5 | 2.0 | 0.5 | 3.0 | 0.5 | 2.5 |
| H. sativum | 1.0 | 3.0 | 0.5 | 2.5 | 1.0 | 2.0 | 0.8 | 2.5 |
| H. sativum | 1.0 | 2.0 | 0.5 | 3.0 | 0.5 | 2.5 | 0.7 | 2.2 |
| Fusarium sp. | 0.5 | 2.0 | 1.0 | 2.0 | 0.5 | 2.5 | 0.7 | 2.2 |
| Curvularia sp. | 1.0 | 1.5 | 0.5 | 1.5 | 1.0 | 3.0 | 0.8 | 2.0 |
| Fusarium sp. | 1.0 | 1.0 | 1.0 | 2.0 | 0.5 | 1.5 | 0.8 | 1.5 |
| Penicillium sp. | 1.0 | 1.5 | 0.5 | 1.5 | 1.0 | 1.5 | 0.8 | 1.5 |
| Mucor sp. | 0.5 | 1.0 | 0.5 | 1.5 | 1.0 | 1.5 | 0.8 | 1.3 |
| Fusarium sp. | 0.5 | 0.5 | 1.0 | 1.0 | 0.5 | 2.5 | 0.7 | 1.3 |
| Mucor sp. | 0.5 | 1.0 | 0.5 | 1.0 | 0.5 | 1.0 | 0.5 | 1.2 |
| Fusarium sp. | 0.5 | 1.0 | 0.5 | 1.5 | 1.0 | 1.0 | 0.7 | 1.2 |

[a] R = resistant inbred, S = susceptible inbred.

[b] Figures 1.0, 2.0, 3.0 and 4.0 equal respectively 25, 50, 75 and 100% rot in inoculated internodes.

the surrounding parenchymatous tissue. This was particularly true with F. moniliforme. Two isolates of H. sativum and two unidentified Fusarium isolates caused approximately the same amount of rot as G. zeae. The isolate of Curvularia also caused a considerable amount of rot. Other organisms including certain species of Fusarium, Mucor and Penicillium produced considerably less rot in the susceptible inbred than the other organisms tested. There was no apparent difference among the organisms in their ability to cause rotting in the resistant inbred. This corn line appeared to be quite resistant to all the organisms tested.

## DISCUSSION

The data in Table 1 show that organisms not commonly thought to be associated with the stalk rot disease were as pathogenic or in some cases more pathogenic than the isolates of G. zeae. Trichoderma spp. have been listed among isolated stalk rot organisms by several workers and have been suspected of being a cause of stalk rot. In these studies, Trichoderma was readily isolated and shown to cause considerable rot in the susceptible inbred. F. moniliforme and G. zeae have long been known to cause stalk rots of corn. Studies have indicated that the majority of the Helminthosporium species isolated from stalk rotted corn were H carbonum (2). From this study it would appear that H. sativum could be a factor in the stalk rot complex. Although only one isolate of Curvularia sp. was used, the results indicate that this particular isolate has the ability to cause rot under these conditions. Of the Fusarium organisms tested, two appeared quite pathogenic and in some instances progressed through vascular strands into the adjacent internodes. These produced little rot in surrounding parenchyma cells. Although some selected species of Fusarium, Mucor and Penicillium produced more rot in the susceptible line than in the resistant, visual observations indicated that the rot incited by these organisms was substantially less than the rot caused by the other organisms tested.

From the results of this study it would appear that several organisms have the ability to cause a rot of corn stalks. However, it is possible that the inoculation technique used in this experiment is particularly favorable for the initiation of rot, regardless of the organism introduced. If so the results obtained with this technique are not necessarily indicative of the susceptibility of corn plants to suspected stalk rotting organisms under natural conditions. The apparent differences in the ability of fungi to cause rot in the susceptible inbred and the lack of extensive stalk rot in the resistant inbred by these organisms would support the validity of this technique.

The resistant inbred corn line which was selected for its standability showed as much apparent resistance to other fungi tested as it did to G. zeae. Although it would appear that several organisms are involved in the stalk rot complex, this selected inbred apparently is equally resistant to all of them.

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# NEMATODE INDUCED GALLS FOUND ON THE ROOTS OF SEA LYME GRASS FROM ICELAND

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Numerous individuals of various stages of the plant parasitic nematode Ditylenchus radicicola (Greeff, 1872) Filipjev, 1936 were isolated from galled roots of sea lyme grass, Elymus arenarius (Fig. 1) from Iceland. This is the first record of the occurrence of this nematode in

FIGURE 1. Galls caused by Ditylenchus radicicola on Elymus arenarius.

Iceland where it was found in a high percentage of the native stands of sea lyme grass examined by the junior author.

The host range and several aspects of the life cycle of this nematode are included in a recent article by Goodey[2].

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[2]Goodey, J. B. 1959. Gall-forming nematodes of grasses in Britain. J. of the Sports Turf Research Institute 10: 1-7.

# FUNGI ISOLATED FROM UNSTORED CORN SEED IN INDIANA IN 1956-1958[1]

John Tuite[2]


## Abstract

Undamaged corn seed taken from a total of 306 fields in Indiana in 3 years, 1956, 1957 and 1958, were cultured on potato-dextrose and malt-salt agar. Cephalosporium acremonium and Fusarium moniliforme were the most common organisms with the former frequent only in certain areas of the State. Other organisms isolated in more than trace amounts but none averaging higher than 4.5% were Gibberella zeae, Alternaria spp., Penicillium spp., Cladosporium sp., Nigrospora oryzae, Mucor spp., and Diplodia maydis. Their incidence varied considerably from year to year.

Of the storage fungi Penicillium spp. were the most common with the Aspergillus-flavus group next and members of A. glaucus, A. niger, A. ochraceus and A. versicolor rarely isolated.

Helminthosporium carbonum and H. maydis were isolated from a few seeds.


There are many reports on the numbers and kinds of fungi isolated from corn seed but most workers used media unsuited for the isolation of certain species of storage molds, for example, members of Aspergillus glaucus group. This study was made to determine the fungi, particularly the storage molds, present in dent corn seed prior to harvest in Indiana. A knowledge of the microflora of unstored corn should aid in the microbiological evaluation of stored corn and perhaps give information on the epidemiology of certain corn diseases.

## MATERIALS AND METHODS

Ten undamaged ears were collected from each of 306 fields in 18 to 20 counties located in southwest, north-central and northwest Indiana in October (about the time of normal harvest). In one year additional samples were collected late into winter. In 1956 and 1957, ears obviously high in moisture were dried at 105° F. In 1958 samples were dried without heat, shelled, mixed in a Boerner divider, and stored at 40° until plated. The seeds were submerged in 5% NaClO for 1 minute, rinsed twice with sterile water, and then 100 seeds were placed on acidified potato-dextrose agar (APDA) and 50 seeds on malt agar containing 7.5% NaCl (MS).

## RESULTS

A treatment of 5% NaClO for 1 minute eliminated most of moderate amounts of external inoculum (1 moldy seed bearing fruiting heads of four storage fungi mixed with 200 noninfested seed). It yielded, however, slightly less amounts of seed infected with A. glaucus as compared with 1% NaClO for 1 minute; the latter treatment and other treatments tried were ineffective in eliminating moderate amounts of inoculum. Consistent numbers and kinds of fungi were obtained when portions of the same samples were plated by different operators, but sometimes considerable variation occurred between samples taken from different places in the same field. Thus it is difficult to appraise the fungus content of corn seed of individual fields by this procedure.

The numbers and kinds of storage fungi isolated on MS and APDA agar (Table 1) indicate some invasion by Penicillium spp. but little by Aspergillus. Of the Penicillia, P. oxalicum was common; however other species were not identified.

The prevalent fungi isolated on APDA are given in Table 2, and their geographical distribution by zones in Tables 3, 4, and 5. The greater numbers of fungi in 1956 and 1957 appeared to be associated with lateness of the crop; most fungi were found in 1957 when corn in most cases was too wet for cribbing. The excessive rainfall in the north-central region at harvest time apparently encouraged Gibberella zeae, Alternaria spp., Nigrospora oryzae, and

[1]Journal Paper Number 1710, Purdue University Agricultural Experiment Station.

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Table 1. Prevalence of storage fungi[a] found in samples of corn seed obtained prior to harvest in 1956, 1957 and 1958 as determined by culturing seed on malt-salt agar and PDA.

| Fungus | Average % of seeds infected | | | High single amount (%) | | |
|---|---|---|---|---|---|---|
| | 1956 | 1957 | 1958 | 1956 | 1957 | 1958 |
| Penicillium spp.[a] | 2.9 | 3.3 | 0.2 | 38 | 22 | 9 |
| Aspergillus glaucus group | 0.1 | .09 | 0 | 6 | 4 | 0 |
| A. flavus group | 0.6 | .09 | .02 | 22 | 8 | 2 |
| A. spp.[b] | .14 | .09 | 0 | 4 | 4 | 0 |

[a]Includes all species of Penicillium, some species do not ordinarily grow in stored corn; the higher amount from the two media is given here.
[b]Includes species of the A. ochraceus, A. niger, and A. versicolor groups.

Table 2. Prevalence of fungi found in samples of corn seed obtained prior to harvest in 1956, 1957 and 1958 as determined by culturing surface disinfected[a] seed on potato-dextrose agar.

| Fungus | Average % seed infected | | |
|---|---|---|---|
| | 1956[b] | 1957[c] | 1958[d] |
| Fusarium moniliforme | 9.0 | 13.3 | 2.2 |
| Cephalosporium acremonium | 15.5 | 10.5 | 7.9 |
| Gibberella zeae | 0.2 | 4.5 | 2.7 |
| Alternaria spp. | .03 | 3.3 | 0.8 |
| Penicillium spp. | 1.9 | 3.3 | 0.2 |
| Cladosporium spp. | 0.1 | 2.0 | 0.2 |
| Nigrospora oryzae | 0.8 | 1.5 | 3.6 |
| Mucor spp. | 0.3 | 0.9 | .07 |
| Diplodia maydis | 0.4 | 0.3 | 0.3 |

[a]100 seed submerged at 5% NaClO for 1 minute.
[b]109 samples examined in 1956.
[c]117 samples examined in 1957.
[d] 82 samples examined in 1958.

Table 3. Distribution and prevalence of fungi found in samples of corn seed obtained prior to harvest in 1956 as determined by culturing surface disinfected seed on potato-dextrose agar.

| Fungus | Average % seed infected | | | |
|---|---|---|---|---|
| | southwestern[a] counties | north central[b] | north west[c] | other[d] |
| Cephalosporium acremonium | 31.4 | 4.1 | 1.6 | 18.6 |
| Fusarium moniliforme | 13.9 | 8.2 | 4.7 | 14.7 |
| Penicillium spp. | 2.3 | 1.5 | 0.8 | 2.1 |
| Nigrospora oryzae | 0.4 | 0.1 | 0.9 | 1.3 |
| Gibberella zeae | 0.1 | 0.3 | 0 | 0.7 |
| Diplodia maydis | 0 | 0.01 | 2.3 | 1.5 |

[a]Includes 27 samples from 5 counties.
[b]Includes 42 samples from 9 counties.
[c]Includes 10 samples from 3 counties.
[d]Includes 1 sample from 1 county.

Cladosporium sp. (Table 4). Samples (results not included in table) collected after early frosts in 1957 had as much as 40 to 50% of the seeds infected with Cladosporium sp., with most of the kernels showing patches of mycelium on their sides. In 1958, preponderance of these four organisms in the southwest (Table 5) again appeared to be associated with the heavy rains that occurred in late July and August.

Cephalosporium acremonium was most common but not widely distributed; it was prevalent primarily in the southwestern counties (Tables 3, 4, 5). It is rare in corn seed in most States (10), common in a few (6, 9), and almost nonexistent in damaged kernels (4). It can be

Table 4. Distribution and prevalence of fungi found in samples of corn prior to harvest in 1957 as determined by culturing surface disinfected seed[a] on potato-dextrose agar.

| Fungus | Average % seed infected: southwestern[b] counties | north central[c] | north west[d] | other[e] |
|---|---|---|---|---|
| Cephalosporium acremonium | 18.7 | 1.5 | 0 | 1.8 |
| Fusarium moniliforme | 11.5 | 11.9 | 6.0 | 3.4 |
| Nigrospora oryzae | 0.8 | 5.2 | 1.0 | 0.8 |
| Gibberella zeae | 1.8 | 4.4 | 3.5 | 1.3 |
| Alternaria spp. | .06 | 13.2 | 5.5 | 0.2 |
| Cladosporium sp. | 0.3 | 2.9 | 3.7 | 0.2 |
| Penicillium spp. | 3.2 | 2.2 | 1.7 | 1.0 |
| Diplodia maydis | 0.9 | 0 | 0 | 0 |

[a]100 seed submerged in 5% NaClO for 1 minute rinsed twice in sterile water.
[b]Includes 42 samples from 6 counties.
[c]Includes 22 samples from 9 counties.
[d]Includes 4 samples from 2 counties.
[e]Includes 8 samples from 3 counties.

Table 5. Distribution and prevalence of fungi found in samples of corn seed obtained prior to harvest in 1958 as determined by plating surface disinfected seed on APDA.

| Fungus | Average % seed infected: southwestern[a] counties | north central[b] | north west[c] | other[d] |
|---|---|---|---|---|
| Cephalosporium acremonium | 31.7[e] | 2.7 | 0.6 | 8.0 |
| Gibberella zeae | 5.2 | 1.2 | 0.6 | 5.4 |
| Nigrospora oryzae | 4.5 | 2.1 | 1.4 | 7.0 |
| Fusarium moniliforme | 5.2 | 1.2 | 0.3 | 3.7 |
| Alternaria spp. | 1.7 | 0.1 | 0.2 | 2.7 |
| Penicillium spp. | 0.2 | 0.1 | 0.1 | 0.1 |
| Diplodia maydis | 0.5 | 0.1 | 0 | 0 |
| Cladosporium spp. | 0.3 | 0.2 | 0.1 | 0.1 |

[a]Includes 13 samples from 5 counties.
[b]Includes 44 samples from 9 counties.
[c]Includes 14 samples from 3 counties.
[d]Includes 13 samples from 4 counties.
[e]Average percentage of infected seed.

missed as it usually grows slowly on APDA. Whether C. acremonium is an aggressive parasite is somewhat controversial, but it is generally believed not to be (1, 3, 8). To test whether black bundle, nubbins and barren stalks arise from infected seed, six lots containing 42 to 81% of C. acremonium were planted in 1957 along with four lots of seed relatively free of internal fungi. Practically all plants appeared healthy with less than 2% of the plants of both groups showing barren stalks, and of the barren stalks less than half coming from Cephalosporium-infected seed and 1/5 from clean seed had black bundles. This lack of obvious disease from kernels infected with Cephalosporium substantiates a recent report (1).

Species identity of the few isolates of Helminthosporium obtained were confirmed by inoculation tests on inbred lines of corn[3]. H. carbonum and H. maydis, both known to attack seed (12), were isolated but H. turcicum was not, although there was considerable northern corn leaf blight in 1957.

## DISCUSSION AND CONCLUSIONS

Aspergilli are uncommon to corn seed in Indiana, prior to harvest. Penicillia occur more frequently but are probably of less importance in commercial storage, because of the inability

[3]Identifications kindly made by Dr. A. J. Ullstrup.

of most species to grow competitively at moisture contents below 16 to 17% (5). The prevalence of C. acremonium and Fusarium moniliforme in seed is probably of little significance in disease outbreaks because of the lack of aggressive pathogenic ability of C. acremonium and the ubiquity of F. moniliforme. C. acremonium (2) and F. moniliforme (11), with their apparent ability to grow at low oxygen and high $CO_2$ tensions, however, may be of potential significance in the hermetic storage of high moisture corn. Seed infection by the majority of the organisms isolated is probably of little importance in epidemiology (6) but Helminthosporia might be introduced into new areas by apparently sound seed.

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# CONTROL OF HEAD SMUT IN RESCUE GRASS[1]

E. S. Luttrell and J. P. Craigmiles[2]

## Summary

Head smut (Ustilago bullata Berk.) often causes severe infection of rescue grass (Bromus willdenovii) in first year stands. The incidence of infection in the same stands decreases in subsequent years because conditions of natural reseeding are less favorable for survival of the fungus. Infection causes direct losses in seed which are of obvious importance in the production of seed crops and in natural reseeding. Smut infection also reduces forage yields. Varieties of rescue grass resistant to smut are available. However, choice of a variety may be based on agronomic characteristics since seed treatment controls the disease.

Under proper management, rescue grass (Bromus willdenovii) is sufficiently persistent in the Southeast to furnish a permanent or semi-permanent winter grazing crop of desirable nutritional quality (3). Although this grass is classed as an annual, many of the plants persist through the summer, and it reseeds readily. Second year stands, therefore, usually furnish earlier and more abundant fall grazing than first year plantings. Head smut (Ustilago bullata Berk.) commonly causes heavy infection of rescue grass in first year plantings, but the disease is markedly reduced in the same plantings in succeeding years. Consequently, studies were made to determine the behavior and importance of this disease and to evaluate seed treatment and the use of disease resistant varieties as methods of control.

## SEED TREATMENT

Seed treatment tests were conducted at the Georgia Experiment Station in 3 years from 1955-1958. Seed of Chapel Hill rescue grass artificially inoculated with smut spores was treated with the following materials at the indicated rates: N-(ethylmercuri)-p-toluene sulfonanilide 7.7% (Ceresan M -- 1/2 ounce/bushel), methylmercury dicyandiamide 2.2% (Panogen 15 -- 3/4 fluid ounce/bushel), tetramethylthiuram disulfide (thiram 75%) (Arasan SF-X slurry -- 6 ounces/100 pounds), and 2,3-dichloro-1,4-naphthoquinone (dichlone 50%) (Phygon -- 6 ounces/100 pounds). Inoculated, untreated seed served as a control. Plantings were made between October 1 and November 1 at the rate of 200 seed per row in 1-row plots 20 feet long in a randomized block replicated five times. The number of seedlings per row was counted approximately 1 month after planting. The following May, after the number of healthy and smutted heads in each row was counted, seed was harvested from all rows.

Results are in Table 1. Seed treatment had little effect on stands, a result in agreement with those of previous studies on other forage grasses (2). Ceresan, Panogen, and dichlone gave complete control of smut, thiram only partial control.

## RESISTANT VARIETIES

Lots of seed of six varieties of rescue grass, including a selection made at the Georgia Experiment Station by J. M. Elrod, were inoculated separately with six smut collections obtained from H. W. Johnson (1) and planted in 1-row plots in a randomized block replicated three times. The planting was made on October 9, 1956 and head counts were taken on May 28, 1957. Results are in Table 2. The low percentage of infected heads in plants from uninoculated of Chapel Hill and Texas 46 indicates that seed of these two varieties was slightly contaminated with smut before it was inoculated. However, additional infection in each of these varieties was caused by each collection of smut. Chapel Hill proved highly susceptible and Texas 46 moderately susceptible. Lamont, Nakuru, Prairie, and the Georgia selection appeared highly resistant.

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[1]Journal Series Paper No. 391, Georgia Experiment Station, Experiment, Georgia.
[2]Plant Pathologist and Agronomist, respectively, Georgia Experiment Station.

Table 1. Effect of seed treatment on stands, head smut infection, and seed yields of Chapel Hill rescue grass.

| Treatment | Results in mean per plot | | | |
|---|---|---|---|---|
| | % seedling stands | total no. heads | % heads smutted | seed yield (grams) |
| Control | 43 | 213 | 79 | 62 |
| Ceresan M | 44 | 202 | 2 | 147 |
| Panogen | 43 | 239 | 0 | 173 |
| Arasan (thiram) | 47 | 225 | 38 | 130 |
| Phygon (dichlone) | 45 | 263 | 0 | 191 |

Table 2. Resistance of rescue grass varieties to head smut.

| Variety | Mean percentage of smutted heads per plot | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Uninoculated | Inoculated with smut collection[a] | | | | | | |
| | | 1 | 2 | 3 | 4 | 5 | 6 | Mean |
| Chapel Hill | 14 | 99 | 87 | 95 | 96 | 96 | 63 | 89 |
| Texas 46 | 6 | 32 | 15 | 34 | 32 | 13 | 48 | 29 |
| Georgia selection | 0 | 0 | 0 | 2 | 0 | 0 | 0 | tr |
| Nakuru | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Prairie | 0 | tr | 0 | tr | 0 | 0 | tr | tr |
| Lamont | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

[a] Smut collections from H. W. Johnson: 1 - Louisiana, 2 - Alabama, 3 - Texas, 4 - Georgia, 5 - Stoneville, Miss., 6 - Raymont, Miss.

## EFFECT OF SMUT INFECTION ON RESCUE GRASS

Data in Table 1 indicate that infection with smut has little effect on emergence and early survival of seedlings since stands 1 month after planting were essentially the same in plots of healthy plants from treated seed and in plots from untreated seed in which high percentages of the plants were infected.

To determine the possible effect of infection on forage yields, a test consisting of 10 replications of paired 5-row plots 10 feet long was designed in 1958. The plots were planted at the rate of 4 g of seed per row on October 8. One plot in each replicate was planted with seed treated with Panogen, the other with untreated seed heavily inoculated with smut spores. Head counts the following spring showed no infection in plots from treated seed and 93% infection in plots from untreated, inoculated seed. A single harvest was made on May 5, 1959. The heads were cut separately by hand, and the plots were then mowed for forage yields. Results in mean pounds of dry weight per plot were as follows: from healthy plants, heads - 1.09, forage - 2.48, total - 3.57; from infected plants, heads - 0.66, forage - 1.55, total - 2.21. The percentage reduction in yield from smut infected plants was 39.4 for heads, 37.5 for forage, and 38.0 for the combined total of heads and forage.

Losses in seed yields are directly related to infection since all seed in smutted heads are destroyed. Reductions in seed yields from plots containing smutted plants as recorded in Table 1 are merely suggestive since the yields would have been much lower if the seed had been thoroughly recleaned. Such seed was practically worthless.

## PERSISTENCE OF SMUT

Two tests were conducted to compare the incidence of smut infection in newly planted stands and in naturally reseeded stands. In the spring of 1956 four 20 x 20 foot split plots were laid out. Seed harvested from plots showing 22% infection with smut was divided in half. Half of the seed was divided into four lots which were broadcast over four of the half plots on July 6. The rest of the seed was stored over the summer and planted in rows on the remaining four half plots on October 1. On May 28, 1958, in the broadcast plots the mean number of plants per plot was 247 and 0.6% were smutted. In the drilled plots the mean number of plants was 2806 and 56% were smutted. Although far better stands were obtained by drilling seed in the fall, this method increased the incidence of infection from 22 to 56%; whereas under natural conditions on the surface of the soil over the summer smut was nearly eliminated.

On October 1, 1956, randomized blocks of five 6 x 20 foot plots in five replications were planted as follows: A and B - smut-inoculated seed, C - inoculated seed treated with thiram, D - uninoculated, untreated seed, and E - not planted. In the spring of 1957 percentages of smut infection in these plots were as follows: A and B - 93, C -47, and D - 8. Plots A were not disturbed, while in plots B, C, and D all plants were removed after seed had been dispersed. In the fall plots E were planted in rows with seed harvested in the spring from plots with 18% of the heads smutted. On May 26, 1958, all plots were sampled by counting the heads enclosed by a 3 x 3 foot counting frame. The mean number of heads per sample in plots A was 229 and 66% were smutted. Many of the smutted plants survived into the second year, but the introduction of healthy plants from natural reseeding reduced the incidence of smut infection from 93% in the first year to 66% in the second year. Natural reseeding reduced the incidence of infection in plots B, with 159 heads per sample, from 93% to 9%; in plots C, with 256 heads, from 47% to 8%; and in plots D, with 346 heads, from 8% to 1%. In plots E, with 105 heads per sample, infection was increased from 18% to 87% by harvesting seed and replanting in the fall.

## CONCLUSIONS

Infection of rescue grass with head smut is favored by the normal process of harvesting, storing, and replanting seed; and unless preventive measures are taken, this process may result in increasing incidence of infection. In natural reseeding, conditions are less favorable for inoculation of the seed and for survival of the smut fungus, and the incidence of infection decreases in the same stand in succeeding years. Since smut causes direct losses in seed yields, its control is essential in seed production. It also causes appreciable losses in forage which, together with the reduction in amount of seed produced for natural reseeding, should be taken into account in plantings for grazing.

Resistance to smut exists in rescue grass, and a variety combining good agronomic characteristics and smut resistance is available in Lamont. This variety is not immune from infection since Johnson (1) indicated that it was moderately susceptible to the smut race represented by the Louisiana collection. Extensive planting of such a variety might result in the widespread development of populations of smut races capable of attacking it. Since smut can easily be controlled by proper seed treatment, the choice of varieties may be made on the basis of agronomic characters. Seed treatment may be justified only for the purpose of controlling smut. Accessory benefits in the form of stand improvement have not been demonstrated.

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## REDUCTION IN YIELD OF COTTON CAUSED BY DISEASES IN 1960

Compiled by the Cotton Disease Council, Committee on Disease Losses: Harlan E. Smith, Chairman; A. L. Smith, W. E. Cooper

The accompanying tabulated summary of the 1960 Cotton Disease Loss Estimates is the ninth report submitted by the committee on cotton disease losses. The summary has been compiled from 49 State estimates. The accuracy of the estimates is highly reliable because most of the same cooperators have contributed for the last 9 years and, as a consequence, their methods and techniques are becoming uniform. The committee feels that considerable credence can be placed in the report.

Table 1. ESTIMATED REDUCTION IN 1960 COTTON YIELD AS A RESULT OF DISEASE DAMAGE.

| Disease | Calif. | Ariz. | N. Mex. | Tex. | Okla. | Mo. | Ark. | La. | Miss. | Ala. | Ga. | S.C. | N.C. | Tenn. | Bales lost | % loss |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. FUSARIUM WILT *Fusarium vasinfectum* | -- | -- | -- | 0.35 | 0.20 | 3.50 | 2.75 | 3.50 | 2.00 | 1.00 | 8.50 | 0.85 | 1.50 | tr | 211,384 | 1.24 |
| 2. VERTICILLIUM WILT *Verticillium albo-atrum* | 2.30 | 3.90 | 7.00 | 2.03 | 0.80 | 3.50 | 0.75 | 1.00 | 0.70 | tr | -- | -- | 0.50 | 1.50 | 288,986 | 1.69 |
| 3. BACTERIAL BLIGHT *Xanthomonas malvacearum* | tr | tr | 1.50 | 5.64 | 4.00 | 3.50 | 0.75 | tr | 0.25 | 0.03 [0.30] | 1.00 | 1.25 | 0.50 | tr | 387,705 | 2.27 |
| 4. ROOT ROT *Phymatotrichum omnivorum* | tr | 2.59 | tr | 5.22 | 0.50 | -- | tr | tr | -- | -- | -- | -- | -- | -- | 312,703 | 1.83 |
| 5. ANTHRACNOSE BOLL ROT *Glomerella gossypii* | -- | -- | tr | 0.07 | tr | 0.20 | 0.10 | -- | 2.25 | 1.00 | 1.25 | 1.50 | 0.40 | tr | 73,345 | 0.43 |
| 6. SEEDLING DISEASES *Rhizoctonia*, etc. | 2.00 | 1.70 | 2.50 | 2.68 | 4.00 | 1.00 | 2.50 | 5.00 | 3.25 | 12.00 | 4.00 | 3.00 | 5.00 | 3.00 | 555,798 | 3.26 |
| 7. ASCOCHYTA BLIGHT *Ascochyta gossypii* | -- | -- | -- | 0.28 | -- | 0.50 | tr | tr | tr | tr | 0.05 | 0.30 | 0.20 | tr | 20,784 | 0.12 |
| 8. BOLL ROTS *Rhizopus*, etc. | 4.00 | 1.75 | tr | 0.63 | 3.00 | 0.10 | 1.40 | 5.00 | 5.50 | 2.00 | 1.00 | 6.00 | 1.75 | 1.00 | 372,906 | 2.19 |
| 9. ROOT KNOT *Meloidogyne* sp. | 1.80 | 1.50 | 1.20 | 2.04 | 0.50 | 3.50 | 0.35 | 1.00 | 0.50 | 3.00 | 5.00 | 4.65 | 4.00 | tr | 308,979 | 1.81 |
| 10. OTHERS | 1.00 | 2.50[a] | 1.00[b] | 2.10[c] | tr[d] | 5.00 | 0.25[e] | -- | tr[f] | -- | 0.50 | 0.85 | 0.30 | tr | 204,042 | 1.19 |
| TOTAL PERCENT LOSS | 11.10 | 13.94 | 13.20 | 21.04 | 13.00 | 20.80 | 8.85 | 15.50 | 14.45 | 19.30 | 21.30 | 18.40 | 14.15 | 5.50 | | 16.03 |
| TOTAL BALES LOST IN HUNDREDS | 2,426 | 1,352 | 441 | 11,159 | 687 | 1,234 | 1,310 | 926 | 2,618 | 1,817 | 1,367 | 936 | 387 | 343 | 27,003[h] | |
| YIELD IN THOUSAND BALES 1960[g] | 1,950 | 835 | 290 | 4,350 | 460 | 470 | 1,350 | 505 | 1,550 | 760 | 505 | 415 | 235 | 590 | | |

[a]Rust (*P. cacabata*, crazy top and others [b]Rust (*P. cacabata*) [c]Largely Pseudomonas wilt [d]Pseudomonas wilt [e]Minor leaf spot
[f]Pseudomonas wilt [g]Dec. 1, 1960 USDA, AMS Estimate [h]In hundreds

COTTON DISEASE COUNCIL, COMMITTEE ON DISEASE LOSSES

# SOURCES, TRANSMISSION, SYMPTOMATOLOGY, AND DISTRIBUTION OF WHEAT STREAK MOSAIC VIRUS IN TEXAS[1]

Lee J. Ashworth, Jr. and Maurice C. Futrell[2]

## Summary

This is the first report concerning the presence of wheat streak mosaic virus disease in Texas. The virus was isolated from native grasses prior to general emergence of fall-planted wheat in areas where no volunteer wheat was found. For that reason, the disease is not believed to be new to the State. During the 1959-60 crop year wheat streak mosaic was found to be generally distributed throughout the Panhandle or High Plains region but it was not found in the north-central or west-central wheat areas. Partial sterility of heads of infected plants was a common field expression of the disease in Texas.

## INTRODUCTION

Wheat streak mosaic, a virus disease, was not observed in Texas until 1959 although its vector, Aceria tulipae (Keifer), has been found on wheat on several occasions (2, 3). The disease was identified from specimens of wheat collected in the northeastern section of the Texas Panhandle by Young, McKinney, and Bellingham[3] in late May 1959, and by the authors in the vicinities of Amarillo and Hereford in early June 1959. No estimates of damage to the 1959 wheat crop could be made because of the late date the disease was recognized. A study of the development and distribution of the disease in the major wheat-producing areas of the State was made during the 1959-60 season; the results appear in this paper.

## METHODS

Four surveys were made in the major wheat-producing belt of the State during the 1959-60 crop season. They were made during the weeks of September 7, November 9, April 11, and May 28. Specimens were frozen before extraction and expressed sap was filtered through qualitative filter paper prior to inoculation as a precaution against the possibility of contaminating the greenhouse with mites.

Identification of manually transmissible virus was based upon symptom expression and incubation period together with mite transmissibility from the wheat specimens that were collected in the fall of 1959. During the spring of 1960 identification was based upon field association of A. tulipae with suspected infected plants and positive mechanical transmission of the virus to wheat in the greenhouse. Several collections of mites, including A. tulipae and A. slykhuisii Hall, were identified by H. H. Keifer[4]. Identifications of subsequent mite collections were made through comparisons with positively identified specimens. F. W. Gould[5] identified all native grass specimens.

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[1]Cooperative investigations of the Department of Plant Physiology and Pathology, Texas Agricultural Experiment Station, College Station and the Crops Research Division, Agricultural Research Service, United States Department of Agriculture. Technical Article 3657 Texas Agricultural Experiment Station.

[2]Plant Pathologists, Texas Agricultural Experiment Station and Crops Research Division, Agricultural Research Service, United States Department of Agriculture, respectively, College Station, Texas.

[3]Streak-mosaic wheat plants were collected from several fields near Perryton, Texas in the northeastern part of the Panhandle region May 20, 1959 by H. C. Young, R. C. Bellingham, and H. H. McKinney. Studies conducted with three separate collections by McKinney, at the U. S. Plant Industry Station, Beltsville, Maryland, indicated that wheat-streak mosaic virus was the causal agent and that the three collections resembled the one described from California by Houston and Oswald (4) in 1952. Virus from each collection was sent to Myron K. Brakke at the Nebraska Agricultural Experiment Station. In a letter to McKinney dated August 11, 1959 Brakke reported that all collections gave a typical wheat-streak mosaic virus zone after density gradient centrifugation and gave a positive reaction against wheat-streak mosaic antiserum.

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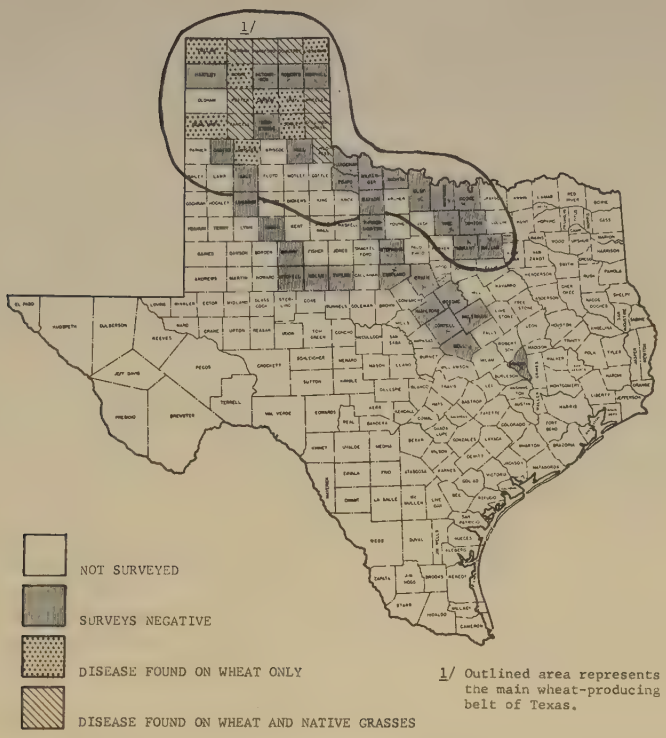
1/
NOT SURVEYED
SURVEYS NEGATIVE
DISEASE FOUND ON WHEAT ONLY
DISEASE FOUND ON WHEAT AND NATIVE GRASSES
1/ Outlined area represents the main wheat-producing belt of Texas.

FIGURE 1. Distribution of wheat streak mosaic in Texas during the 1959-1960 crop season.

FIGURE 2. Wheat streak mosaic induced floret sterility symptoms in field-grown Concho wheat. Left, normal head; right, five heads from infected plants.

In one instance limited antigenic analysis was made of a wheat virus isolate suspected of being barley stripe mosaic virus. An agar gel double diffusion technique, suggested by Moorhead (6) and similar to the method described by Bjorklund (1), was used. Commercially produced barley stripe mosaic virus antiserum[6] was tested against the unknown virus from wheat, sap from barley plants that were known to be infected with barley stripe mosaic virus, and normal rabbit serum. Two tests of four replicates using three specimens of the unknown virus were made.

## RESULTS

A search for wheat streak mosaic in native grasses and volunteer wheat was made in early September 1959. Emphasis was placed upon grasses known to be hosts of the virus. Figure 1 shows the area involved in the first and subsequent surveys. In the areas surveyed in September, volunteer wheat was emerging with seeded wheat. Seedling wheat was found only in the upper two tiers of counties of the Panhandle at that time. No virus-like symptoms were observed in wheat, but mosaic symptoms were common in several species of grasses that were generally distributed throughout the Panhandle. Panicum capillare and Setaria viridis were the most common grasses with mosaic symptoms, but Cenchrus pauciflorus, Echinochloa crusgalli, and Eragrostis cilianensis were less commonly infected but were generally distributed in that region. No mosaic symptoms were found in grasses in either the north-central or the west-central parts of the State.

The responses of wheat, barley, and oats to some of the grass and wheat virus isolates appear in Table 1. In agreement with McKinney's report (5), the incubation period of the virus

Table 1. Host sources, transmission, and symptomatology of some collections of wheat streak mosaic virus specimens on Wichita wheat (C.I. 11952), Atlas barley (C.I. 4118), and New Nortex oats (C.I. 3422) collected in September and November, 1959.

| Source | Aceria tulipae association with field specimen | Transmission by A. tulipae in greenhouse test | Wichita wheat: incubation period[a] | Wichita wheat: type of symptoms[b] | Atlas barley: incubation period | Atlas barley: type of symptoms | New Nortex oats: incubation period | New Nortex oats: type of symptoms |
|---|---|---|---|---|---|---|---|---|
| Setaria viridis | Negative | Positive | 8 | 2 | 23 | 3 | 14 | 1 |
| do. | do. | do. | 7 | 2 | 19 | 3 | 12 | 1 |
| Panicum capillare | do. | do. | 7 | 2 | 18 | 3 | 14 | 1 |
| do. | do. | do. | 8 | 2 | 19 | 3 | 14 | 1 |
| do. | do. | do. | 9 | 2 | Negative | Negative | 10 | 1 |
| do. | do. | do. | 10 | 1 | do. | do. | -- | - |
| Winter wheat | Positive | do. | 7 | 2 | 20 | 3 | 14 | 1 |
| do. | Negative | do. | 7 | 1 | 13 | 2 | 14 | 1 |
| do. | do. | do. | 11 | 1 | Negative | Negative | Negative | Negative |
| Panicum sp. | Positive | Negative | 10 | 1 | do. | do. | -- | - |
| Cenchrus pauciflorus | Negative | do. | 10 | 1 | do. | do. | -- | - |
| Eragrostis cilianensis | do. | do. | 10 | 1 | do. | do. | -- | - |
| Echinochloa crusgalli | do. | do. | 9 | 2 | 18 | 3 | 14 | 1 |
| Winter wheat | do. | do. | 9 | 1 | 20 | 2 | 14 | 1 |
| do. | do. | do. | 12 | 2 | 20 | 3 | 14 | 1 |

[a]Incubation period in days.

[b]1, systemic mottle only; 2, severe systemic mottle and leaf distortion; 3, broad chlorotic streaks followed by systemic mottle.

isolates was usually shortest in wheat, 7 to 12 days, and longest in barley, 13 to 23 days. The incubation period was also a few days longer in oats, 10 to 14 days, than in wheat. Some isolates of the virus induced only a systemic streak-mosaic on Wichita (C.I. 11952) wheat, where-

[6]Microbiological Associates, Inc., 4813 Bethesda Avenue, Washington 14, D.C.

as others caused a severe systemic mottle together with leaf distortion as described by Slykhuis (8) and Houston and Oswald (4). Except for two virus strains, only those that caused leaf distortion in wheat (Table 1) caused symptoms in Atlas (C.I. 4118) barley. Such strains caused symptoms in barley which appeared to be identical to those found in Atsel barley by McKinney (5). In barley, symptoms were first evident as broad linear bands that lengthened acropetally and eventually extended over the entire length of the leaf. Some leaves died at that point but the rest of the leaves on the plant and new ones then developed mild to severe streak mosaic symptoms. The two strains that were mild on wheat but were transmissible to barley caused severe mottle symptoms and leaf distortion to that plant. New Nortex (C.I. 3422) oats exhibited only mild mosaic symptoms in response to infection by the various wheat virus isolates.

In September, Eriophyid mites were found only on Panicum species; A. tulipae was found only once and then on a tall species of Panicum. In contrast, nearly all collections of P. capillare were heavily infested with A. slykuisii. Two attempts to culture this mite by incubating eggs on wheat seedlings failed under conditions where A. tulipae developed. Del Rosario (7) was not able to rear the mite on wheat and it did not transmit wheat streak mosaic virus to wheat in her tests.

By November wheat streak mosaic was found on wheat throughout the Panhandle. It was most common in the western half of the area where winter wheat was generally planted quite early. Only traces occurred in the east side of the Panhandle where wheat was generally planted quite late in the fall. The disease was not found on wheat in either the north-central or west-central wheat-producing areas in November. A. tulipae occurred wherever the disease was found in November. Mite populations were high by April and appeared to be maximum by early June.

Some evidence that late-fall or early-spring infections of winter wheat by wheat streak mosaic virus can severely reduce grain yield was obtained. A field of irrigated Concho (C.I. 12517) wheat planted adjacent to a volunteer field and which emerged the week of October 5 was uniformly infected with the virus in early June. Nearly all plants showed severe yellow streak mosaic symptoms and 10 to 20% of the heads of infected plants were partially or wholly sterile (Fig. 2). The field yielded slightly less than 30 bushels/acre. A. tulipae was not found except at the margin of the field adjacent to volunteer wheat. For that reason the disease was believed at first to be due to barley stripe mosaic virus. However, host-range studies, a negative test for seed transmission, and a negative reaction to barley stripe mosaic virus antiserum indicated it to be due to wheat streak mosaic virus. Partially sterile heads of wheat in other fields infected with the virus were common, particularly in the irrigated wheat area of Dallam, Sherman, and Moore counties. A similar observation was made on manually inoculated Wichita wheat in the greenhouse (Table 2). Each of 13 isolates was inoculated on four 2-week-old plants. These results agree with observations made in the greenhouse by Slykhuis (8) and Houston and Oswald (4).

Table 2. Influence of wheat streak mosaic virus upon development of partially or completely sterile heads of Wichita wheat under greenhouse conditions.

| Treatment of plants | Number of plants | Number of heads | Partially and completely sterile heads | |
|---|---|---|---|---|
| | | | number | % |
| Not inoculated | 12 | 34 | 5 | 14.7 |
| Inoculated[a] | 52 | 66 | 39 | 59.1 |

[a]Four isolates completely inhibited flowering.

## DISCUSSION

Wheat growers of the Panhandle area of Texas suffered severe yield losses during the 1958-1959 growing season. Wheat streak mosaic virus was suspected to be a major cause of losses but because of the stage of maturity of wheat in the region, symptoms were difficult to detect and the virus was isolated and identified in only a few instances. Therefore, the role the virus played during the 1958-1959 season is highly questionable.

A virus was mechanically transmitted from seeded and volunteer wheat to healthy wheat seedlings 54 times and from 6 species of native grasses 12 times during the course of the 1959-1960 growing season. Transmission tests with A. tulipae were attempted only once for any particular virus isolate, so little emphasis can be placed upon negative results. It was con-

cluded from host-range studies that the virus in question was wheat streak mosaic. The virus is not believed to be new in Texas because it was found in native grasses prior to general emergence of fall-planted wheat. Native grass hosts of the virus appear important in the development of the disease in Texas although the vector relationships of grass and wheat hosts are poorly understood.

Wheat yields for the State were higher in 1960 than in 1959. Nevertheless, wheat streak mosaic caused substantial losses in Dallam, Sherman, Moore, and Deaf Smith counties, with less damage being done in Potter, Carson, Randall, and Gray counties. Scattered individual fields were seriously affected by the virus in other counties in the Panhandle wheat area.

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# THE INCIDENCE OF STEM ROTS IN TOBACCO TRANSPLANTS IN RELATION TO WIREWORM INJURY

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## Summary

In field experiments in North Carolina, where insecticides were being evaluated for control of the wireworm, Conoderus vespertinus (F.), a high positive correlation was found between the incidence of two stem-rot diseases of tobacco transplants, caused by Sclerotium rolfsii and Pythium spp., and the amount of wireworm damage. It is concluded that certain stem-rot pathogens readily enter tobacco transplants through wounds caused by wireworms.

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The wireworm, Conoderus vespertinus (F.), is the most destructive insect pest of tobacco transplants in North Carolina. Although the entire plant is subject to attack, most conspicuous damage involves the stems at or near the soil surface where these pests either bore into and tunnel in the pith or feed in the outer cortical tissues. In severe cases the stems may be girdled or weakened to such an extent that the plants are killed. Such injuries might provide infection courts for stem-rot pathogens. Both southern stem-rot, caused by Sclerotium rolfsii Sacc., and pythium rot, caused by Pythium spp., are common fungal diseases of tobacco transplants in North Carolina. In field experiments, where insecticides were being evaluated for wireworm control, material was available for studying the relationship between these stem-rot diseases and wireworm damage. The results of the wireworm control phases of this work have been published[2]. This report deals with the stem-rot studies.

## METHODS

According to a pre-season assay, fields selected for the experiments were expected to have a high incidence of wireworm injury. Single row plots 50 to 100 feet in length were replicated four times. Insecticide treatments for wireworm control were applied either as row treatments one week before setting tobacco plants, or in the transplanting water when plants were set. In all check and row-treated plots, the plants were set with nontreated water. About one to two weeks after setting, 20 to 50 plants were pulled from each plot and examined in the laboratory. Plants which had been bored at least 1/2 the diameter of the stem, tunneled at least 1/2 inch, or showed more extensive injury were scored as wireworm injured. No attempt was made to evaluate injury on the roots of the plants. The two diseases were easily diagnosed by their characteristic symptoms. Both pathogens cause local lesions at or near the soil surface. Pythium incites a typical soft rot in which the affected tissues become flaccid and grayish-green in color. S. rolfsii macerates parenchymatous tissues leaving strands of vascular elements in a shredded condition.

## RESULTS AND CONCLUSIONS

The results of the three-years' test are shown in Table 1. These data were analyzed by analyses of variance and covariance[3].

At Clayton-1, in 1958 there was a definite correlation between the incidence of southern stem-rot and wireworm injury. Upon analysis, 86% of the variation in stem-rot was associated with parallel variation in wireworm damage with respect to treatments means and 59% with respect to individual plots means. Such association is highly significant. The Clayton-2 test indicated the same general trend, and the association between wireworms and stem-rot was 91% with respect to treatments means. In both cases, factors other than wireworm damage were also operative. In the 1959 test, a similar trend occurred with southern stem-rot when there was a relatively low incidence of this disease. The results with pythium rot in both 1959 and

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[1]Professor of Plant Pathology and Associate Professors of Entomology, respectively.

[2]Guthrie, F. E., W. E. Splinter, R. L. Rabb, and T. G. Bowery. 1960. Mechanical transplanting of bright leaf tobacco. IV. Tobacco wireworm control with row treatment of insecticides. Tob. Sci. 4: 95-100.

[3]The help of Dr. D. D. Mason, Department of Experimental Statistics, North Carolina State College, Raleigh, North Carolina in making the statistical analyses is herewith acknowledged.

Table 1. Incidence of wireworm and stem-rot damage to tobacco transplants at various locations, 1958-60.

| Location | Year[a] | Treatment: material[d] | dosage (pounds/acre) | application method[b] | % plants injured by wireworms | % plants affected by: Pythium rot | % plants affected by: Southern stem-rot |
|---|---|---|---|---|---|---|---|
| Clayton-1[c] | 1958 | heptachlor | 0.5 | R | 35 | 0 | 21 |
| | | do. | 0.5 | R | 5 | 0 | 8 |
| | | do. | 0.5 | R | 8 | 0 | 11 |
| | | do. | 0.5 | R | 28 | 0 | 19 |
| | | do. | 0.5 | R | 17 | 0 | 22 |
| | | do. | 0.25 | TW | 6 | 0 | 9 |
| | | Nematode DD136 | | R | 71 | 0 | 31 |
| | | do. | | TW | 30 | 0 | 9 |
| | | Am. Cy. 12880 | 0.25 | TW | 29 | 0 | 15 |
| | | Am. Cy. 18706 | 0.25 | TW | 60 | 0 | 22 |
| | | Check | - | - | 76 | 0 | 43 |
| | | | | LSD .05 | 16 | | 14 |
| Clayton-2 | 1958 | Ortho RE 4355 | 0.25 | TW | 58 | 0 | 57 |
| | | ethion | 0.25 | TW | 54 | 0 | 59 |
| | | Thiodan | 0.25 | TW | 48 | 0 | 61 |
| | | Sevin | 0.25 | TW | 16 | 0 | 42 |
| | | Trithion | 0.25 | TW | 42 | 0 | 48 |
| | | Bacillus | 0.90 | TW | 57 | 0 | 62 |
| | | Check | - | - | 69 | 0 | 72 |
| | | | | LSD .05 | 12 | | 14 |
| Clayton | 1959(A) | aldrin | 0.5 | R | 12 | 0 | 0 |
| | | Check | - | - | 57 | 18 | 6 |
| | (B) | aldrin | 0.5 | R | 10 | 1 | 0 |
| | | Check | - | - | 62 | 20 | 7 |
| | | | | LSD .05 | 34 | 15 | 3 |
| Whiteville | 1960 | lindane | 0.04 | TW | 9 | 8 | 0 |
| | | Check | - | - | 54 | 23 | 0 |

[a]Tobacco varieties used: 1958 - N.C. 73; 1959(A) - Hicks, (B) - Coker 187-Hicks; 1960 - Coker 187-Hicks.

[b]R = row application; TW = transplanting water.

[c]See Guthrie, et al.[2] for specific details of the 1958 Clayton-1 test.

[d]heptachlor= 3a,4,5,6,7,8,8-heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene; Nematode DD 136= Accession number of an unnamed nematode; Am. Cy. 12880= O,O-dimethyl S-(N-methylcarbamoylmethyl) phosphorodithioate (=dimethoate); Am. Cy. 18706= O,O-dimethyl S-(N-ethylcarbamoylmethyl) phosphorodithioate; Ortho RE 4355= 1,2-dibromo-2,2-dichloroethyl dimethyl phosphate (=Dibrom); ethion= O,O,O',O'-tetraethyl S,S'-methylenebisphosphorodithioate; Thiodan= 6,7,8,9,10,10-hexachloro-1,5,5a,6,9,9a-hexahydro-6,9-methano-2,4,3-benzodioxathiepin-3-oxide; Sevin= 1-naphthyl methylcarbamate; Trithion= S-(p-chlorophenylthio)methyl O,O-diethyl phosphorodithioate; aldrin= 1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-1,4-endo-exo-5,8-dimethanonaphthalene; lindane= gamma-1,2,3,4,5,6-hexachlorocyclohexane.

1960 were highly significant in showing a positive correlation between the incidence of this disease and wireworm injury.

It is concluded that certain stem-rot pathogens readily enter tobacco transplants through the wounds caused by wireworm feeding.

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# RHYNCHOSPORIUM SCALD, SEVERE DISEASE OF BARLEY IN GUATEMALA

Eugenio Schieber and Astolfo Fumagalli[1]

Rhynchosporium scald on barley (Hordeum vulgare), incited by Rhynchosporium secalis (Oud.) J. J. Davis, is one of the potential limiting factors to the establishment of barley plantings in the highlands of Guatemala. The disease developed in a severe outbreak in the experimental plots at the Labor Ovalle station of the Instituto Agropecuario Nacional, located at an elevation of 7900 feet. Rhynchosporium scald was also found attacking plantations scattered through western Guatemala.

Symptoms on local and introduced varieties correspond to those described by Dickson[2]. Blotches develop on leaves and sheaths, first water-soaked, ovate to irregular in shape. Later lesions show a zonated scald with the centers of a light straw color and brown margins (Fig. 1). Conidia are hyaline, one septate, cylindrical with a short apical beak. Inoculum persists during the dry season on dead leaf tissues and also on sporadical out-of-season plantings in the highlands of Guatemala.

During the rainy season of 1960 only a few varieties introduced from the United States and other countries showed some degree of resistance. A selection of hull-less barley introduced from Utah, U.S.A. rated high in resistance. Forrest showed high susceptibility during the rainy season, but late plantings of this variety in the dry season showed no disease. This later observation indicated that a program of date of planting together with the search for resistance will solve in part the problem of Rhynchosporium scald in Guatemala.

FIGURE 1. Disease symptoms on barley showing the zonated scald.

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[1]Head Plant Protection Department and Head Small Grain Program, respectively.

[2]Dickson, J. G. 1956. Diseases of Field Crops. 2nd Ed. McGraw-Hill Book Co., New York. pp. 49-51.

# CHERRY ROSETTE: ITS NONIDENTITY WITH PFEFFINGERKRANKHEIT AND ITS POSSIBLE AFFINITY WITH STECKLINBURGER DISEASE[1]

R. M. Gilmer

## Summary

Cherry rosette, a serious virus disease of Montmorency cherry in the northeastern United States, is apparently induced by Fulton's virus E, an anomalous strain of necrotic ringspot virus. Although the symptoms of rosette superficially resemble those of Pfeffingerkrankheit in several respects, the causal viruses differ greatly in herbaceous host range and in physical properties and thus do not appear related.

Symptoms of rosette resemble those of Stecklinburger disease closely and the causal viruses possess certain features in common. In the absence of direct comparisons of the two viruses, definite identification is not possible but it appears probable that rosette and Stecklinburger disease are induced either by the same virus or by two very similar viruses.

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Lewis (14) and Adams and Kessler (1) simultaneously reported outbreaks of cherry rosette in Pennsylvania and West Virginia, and Adams and Kessler presented evidence of its virus etiology. These authors concluded, on the basis of similarities in symptomatology, that rosette was identical with Pfeffingerkrankheit (Eckelrader disease) reported by various European workers (3, 15, 16). Pfaeltzer (16) accepted this identification and proposed that the terms Pfeffinger and Eckelrade be suppressed in favor of the term rosette.

Evenhuis, et al. (5) demonstrated the transmission of Pfeffinger virus through soil. Cadman (4) provided general substantiation of soil-transmission of Pfeffinger virus, since he recovered either raspberry ringspot virus or raspberry yellow dwarf virus (arabis mosaic virus) from sweet cherry trees with Pfeffingerkrankheit growing in such diverse areas as Switzerland, Germany, and England. Both of these viruses are known to be soil-borne, and arabis mosaic virus has recently been shown to be transmitted by a nematode, Xiphinema sp. (11, 12).

Thus, the identification of rosette with Pfeffingerkrankheit, if correct, indicated the presence of a serious soil-borne virus in the northeastern United States. Since the writer had observed experimental work on the herbaceous host range and physical properties of Pfeffinger virus in England, Scotland, and the Netherlands in 1958, it seemed highly desirable to conduct similar experiments with rosette virus to determine whether its identification with Pfeffinger virus was correct. Since it was inadvisable to import authentic cultures of Pfeffinger virus, direct comparisons were not made.

## EXPERIMENTATION

Five virus isolates were compared: three isolates of rosette virus from Pennsylvania (201, 202, 203) obtained from Dr. F. H. Lewis; a single isolate of vein-clearing rosette virus (10) indigenous to New York (Cr 1-30); and a representative strain of necrotic ringspot virus (B 7/8).

All five isolates were individually transmitted by bud-inoculation to virus-free seedlings of Prunus mahaleb and P. persica; seedlings were inoculated just prior to budbreak. In most cases symptoms became evident within 15 to 25 days after inoculation.

In mahaleb seedlings, each isolate induced ringspots with necrosis and rosetting which was followed by partial recovery with diminution in symptom severity. Symptoms induced by isolates 201, 203, and CR 1-30 appeared to be initially more severe and somewhat more persistent than those induced by isolates 202 and B 7/8, but there was considerable variation in the severity and persistence of symptoms within an individual group of trees inoculated with the same isolate.

In peach, all five isolates again induced ringspotting, necrosis, and pronounced rosetting, followed by more or less complete recovery. Isolates 201 and CR 1-30 induced a more persistent rosetting and more severe foliage necrosis than the remaining isolates.

In spite of the real or apparent differences in symptom severity and rate of recovery, the reactions in both peach and mahaleb appeared to lie within the range commonly induced by isolates of necrotic ringspot virus.

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[1]Approved by the Director as Journal Paper No. 1251, New York State Agricultural Experiment Station, Geneva.

Each isolate was mechanically transmitted from Montmorency foliage extracts to cucumber seedlings (var. Ohio MR 17). Each of the five isolates induced a severe disease characterized by local lesions on the inoculated cotyledons, severe necrosis of the growing point, and rapid collapse and death of the entire plant. During March and April cotyledonary lesions were usually necrotic, but as daylength increased the lesions tended to become chlorotic rather than necrotic, and infected plants survived for increasing periods of time. The tendency to produce necrotic cotyledonary lesions persisted longer for isolates CR 1-30 and 201 than for the remaining isolates; the representative necrotic ringspot virus isolate B 7/8 appeared to be the least severe in effect.

Each isolate was tested on a herbaceous host range of Cucurbita pepo (Cocozelle squash), C. moschata (Butternut squash), Citrullus vulgaris (Dixie Queen watermelon), Nicotiana tabacum, and Chenopodium amaranticolor. Each isolate readily infected both Cocozelle and Butternut squashes and induced poorly defined cotyledonary lesions followed by death of the growing point. Occasional squash plants produced one (rarely two) true leaves before the growing points became necrotic; such leaves occasionally developed large semi-necrotic areas. Cotyledonary lesions of isolates CR 1-30 and 201 usually tended to become necrotic as the squash cotyledons became senescent.

In Dixie Queen watermelon each isolate induced pin-point (1 mm or less) necrotic flecks on the cotyledons; these flecks were usually few in number and the individual isolates could be recovered from watermelon only with considerable difficulty. The lesions induced by isolate CR 1-30 tended to be slightly larger in size and often became whitish in color.

None of the isolates was transmitted to tobacco or to Chenopodium amaranticolor, or, if transmitted, could not be recovered from inoculated plants of these species. In one trial, isolate CR 1-30 apparently induced a few whitish pin-point flecks on the inoculated leaf of a plant of C. amaranticolor, but if these flecks were actually virus-induced the causal virus could not be recovered.

The thermal inactivation point of each isolate was determined in cucumber sap, partially clarified by low-speed centrifugation to remove particles of plant debris. Cucumber cotyledons were harvested just as the cotyledonary lesions first became evident (usually 4 to 6 days after inoculation) and 1 gram of fresh tissue was triturated in 5 ml phosphate buffer (.06 M, pH 8.3). Samples were heated for the standard 10-minute period in a circulating water bath accurate to ± 1°C and were plunged into ice water immediately after removal from the water bath. Data presented in Table 1 were obtained in two trials.

The infectivity of each isolate after aging in vitro was also determined in two trials. In order to permit comparison with the aging data of Fulton (7), cucumber sap at an approximate dilution of 1:25 with .03 M phosphate buffer was aged at a temperature of 20° to 24°C. In a second trial cucumber sap diluted 2:5 with .06 M phosphate buffer and aged at 5° permitted a rough comparison with data of Baumann (2) for Stecklinburger virus. These data are presented in Table 1.

Table 1. Thermal inactivation and aging in vitro tests of four isolates of rosette virus and an isolate of NRSV.

| Isolate | Thermal inactivation point (°C) | Duration of infectivity (hours) | |
|---|---|---|---|
| | | 22° to 24°C | 50°C |
| Rosette 201 | 53-56 | 10 | 12 |
| Rosette 202 | 56 | 12 | 12 |
| Rosette 203 | 56 | 12 | 12 |
| Rosette CR 1-30 | 56 | 8 | 12 |
| NRSV B 7/8 | 53 | 8 | 10 |

None of the isolates remained infective when whole infected cucumber cotyledons were exposed to -20°C for 2 hours.

## DISCUSSION

Although direct comparisons of rosette and Pfeffinger viruses were not made, the host ranges and physical properties of the two viruses are so different that they cannot be considered to be the same virus. The thermal inactivation point of Pfeffinger virus has been reported as 60° to 65°C (10 minutes) by Pfaeltzer (16) and as 65° (10 minutes) by Baumann (2). Baumann

also found that Pfeffinger virus remained infectious slightly more than 50 hours when aged in vitro either at 18° or at 2°.

Pfaeltzer (16) infected tobacco with Pfeffinger virus where the virus induced small white concentric rings or necrotic rings; she also found that Pfeffinger virus induced small chlorotic spots on cucumber cotyledons followed by a general systemic mottle and recovery. Kegler (13), in concluding that Stecklinburger and Pfeffinger viruses were different, reported recovery of Pfeffinger virus from Chenopodium foetidum.

Rosette virus shows pronounced differences. The four isolates tested were inactivated at 53° to 56°C (10 minutes) and none remained infective in vitro for more than 12 hours. None of the rosette isolates were transmitted to tobacco and only one (CR 1-30) doubtfully to C. amaranticolor from which it was not recovered. The symptoms induced in cucumber by the four rosette isolates -- apical necrosis and rapid collapse -- differ from the symptoms induced in cucumber by Pfeffinger virus as reported by Pfaeltzer (16).

All of the rosette isolates induced symptoms in peach and mahaleb seedlings that did not differ appreciably from symptoms induced by a representative strain of necrotic ringspot virus, although, in general, the symptoms of rosette were somewhat more severe. Symptoms induced in cucumber, two species of squash, and watermelon by rosette and necrotic ringspot viruses were likewise very similar. In these respects the four rosette isolates resemble Fulton's virus E (6, 7, 8), which has recently been shown to be related serologically to necrotic ringspot virus (9).

The thermal inactivation points of rosette virus, necrotic ringspot virus, Fulton's virus E, and Stecklinburger virus are in close agreement: rosette virus - 53° to 56°C; necrotic ringspot virus - 53°; virus E - 56° (7); Stecklinburger virus - 57° (2). Fulton has already pointed out the possible affinity of virus E with Stecklinburger virus. In view of the very marked similarity of symptoms in Montmorency and Morello cherry (budblast, leaf enations, dieback) and in cucumber induced by virus E, rosette virus, and Stecklinburger virus and the close agreement in physical properties, these three viruses appear closely related if not actually synonymous.

Adams and Kessler (1) reported that certain Montmorency trees with rosette later developed symptoms of necrotic ringspot, and Fulton (8) found that previous infection with necrotic ringspot virus did not inhibit the development of "shock" symptoms by virus E. These data suggest that in spite of the serological affinity between virus E and necrotic ringspot virus the two viruses are not closely related.

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# A NEW HOST FOR THE CUCURBIT POWDERY MILDEW FUNGUS

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## Abstract

Two plants of turkey mullein, Eremocarpus setigerus, that emerged as weeds in greenhouse plantings of cantaloupe, Cucumis melo, developed powdery mildew following routine inoculation with the cucurbit powdery mildew fungus. The fungus was successfully transferred from the turkey mullein to cantaloupe seedlings grown in the laboratory. This appears to be the second demonstration that a cucurbit powdery mildew fungus collected in southern California is not limited in its host range to the Cucurbitaceae. The certain identification of the cucurbit powdery mildew fungus clone reported here is withheld pending the production of perithecia. Turkey mullein, and perhaps other non-cucurbitaceous weeds, may have roles in the dissemination of the cucurbit powdery mildew fungus in nature.

## INTRODUCTION

Two plants of turkey mullein, Eremocarpus setigerus, (identification verified by Dr. J. M. Tucker, Associate Professor of Botany, University of California, Davis) emerged as weeds in soil used to grow cantaloupe (Cucumis melo) seedlings in the greenhouse for routine inoculations with the powdery mildew fungus. The soil had been purchased from a local nurseryman, who stated that it was obtained from the vicinity of Alpine, California. Robbins, Bellue, and Ball (11) and Munz and Keck (9) stated that turkey mullein is a native of the Pacific Coast States and is common there on disturbed soil below a 2500-foot altitude.

Both the stems and lower leaves of the turkey mullein plants became infected with a powdery mildew fungus. The infection suggested the question: did the plants of turkey mullein become infected with the powdery mildew fungus used for inoculation of the cantaloupe or with a fungus from some other source brought in with the nursery soil?

## HISTORY OF THE FUNGUS CULTURE

The fungus used for the resistance tests of cantaloupe breeding lines was collected from plants of the No. 450 variety of cantaloupes grown in the Imperial Valley of California. It had been screened for virulence on the Powdery Mildew Resistant No. 5 variety (4) and cultured on the No. 45 and No. 450 varieties (4) in the greenhouses at La Jolla for several years. The culture was moderately virulent to the No. 5 variety and very virulent to the No. 45 and No. 450 varieties of Cucumis melo. It was assumed, therefore, to be a clone of Erysiphe cichoracearum DC. race 2 as described by Jagger, Whitaker and Porter (6). The clone was virulently parasitic also to cucumber (C. sativus), squashes and pumpkins (Cucurbita pepo, C. maxima, C. moschata, C. mixta), and some gourds (C. foetidissima, C. andreana). It was moderately virulent to some other gourds (C. lundelliana and C. palmata) but avirulent to watermelons (Citrullus vulgaris) and to wild cucumber (Marah macrocarpa). Whitaker and Pryor (16) reported similar relationships of an earlier clone of cucurbit powdery mildew race 2 to some of those hosts and to certain other cucurbits. Prior to this study neither clone had been known to parasitize plants other than members of the Cucurbitaceae. Neither clone has been observed to produce perithecia on any host in southern California including U. S. Plant Introductions 179260 and 181910 of Cucumis sativus reported by Randall and Menzies (10) to support perithecia of a cucurbit strain of E. cichoracearum in Washington.

## EXPERIMENTAL PROCEDURE AND RESULTS

An attempt was made to infect cantaloupes with the fungus growing on the turkey mullein without the occurrence of contaminating infection by conidia directly from cantaloupes. The cantaloupe plants were removed from a pot that contained one of the turkey mullein seedlings. The pot with only the turkey mullein plant was then placed on a window sill in a laboratory for a month (about twice the conidium-longevity period reported by Yarwood, et al., 18) to allow

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the powdery mildew fungus conidia from the cantaloupe plants to die. After the isolation period cantaloupe seeds were planted in the pot containing the turkey mullein plant. The cantaloupe seedlings were left undisturbed until the first true leaf expanded. Examination with a hand lens showed that they remained free from infection in the still air in the laboratory during this period.

The powdery mildew fungus was removed from the turkey mullein plant with a moistened scalpel, and deposited gently on some of the cotyledons and first leaves of the cantaloupe seedlings. Care was exercised to avoid disturbing the mildew sufficiently to cause inoculations at sites other than those selected for inoculation.

Growth of the powdery mildew fungus was visible at all inoculated sites a week after inoculation. Ten days later the mildew colonies appeared to be identical with those on cantaloupe seedlings in the greenhouse infected with the fungus obtained directly from cantaloupes in the routine resistance tests. The structure of the fungus, including conidium size, appeared to be identical with that of the powdery mildew fungus grown continuously on cantaloupes in the greenhouse. No mildew growth occurred at sites other than those inoculated on the plants in the laboratory during 6 weeks that they were kept under observation. We concluded that 1) the fungus growing on the turkey mullein was capable of infecting cantaloupe, and 2) the turkey mullein was infected with the powdery mildew fungus that was used to inoculate cantaloupes in the greenhouse.

## DISCUSSION

Most of the studies on cucurbit powdery mildew fungi (7, 8, 13, 16 et al.) have been performed with conidial (Oidium) stages. The assignment of these fungi to Erysiphe cichoracearum DC. seems to be based on the comparatively rare reports of perithecia of that species on cucurbitaceous hosts (1, 10, 12). As Salmon (12) stated, "The practice of referring a fungus in its Oidium stage to a certain species of the Erysiphaceae has led ..... to very numerous mistakes, and has made any compilation of a host-index from published records alone utterly misleading."

Blumer (1) reviewed the literature on inoculation experiments with E. cichoracearum and concluded that the species exhibits strong specialization at the host family level. He emphasized host specialization in his concept of the species. Schmitt (13) reported that E. cichoracearum collected from squash appeared to have a host range limited to the Cucurbitaceae. His Michigan isolate failed to produce perithecia and it failed to infect 23 species in six other families. Miller and Barrett (8) reported that Erysiphe cichoracearum cultures from cantaloupe (probably race 1 as described by Jagger, Whitaker and Porter, 6) and from sunflower (Helianthus annuus) failed to cause infection following reciprocal cross-inoculations. Perithecia were produced by the fungus on the sunflower but not by that on the cantaloupe. Miller (7) reported the occurrence of a cucurbit powdery mildew on papaya (Carica papaya) in southern California. Our observations on mildew-free crop plants of various species grown in proximity with mildewed cantaloupes suggest that the clone of the cucurbit powdery mildew fungus reported here is closely related to those reported by Schmitt, Miller, and Miller and Barrett.

Schnathorst, Grogan and Bardin (15) found strains of Erysiphe cichoracearum from lettuce (Lactuca sativa) and wild lettuce (L. serriola) to be parasitic on detached leaves, but not on potted plants, of certain varieties of Cucumis melo and C. sativus. That fungus differed from the clone considered here by its failure to attack potted plants of cucurbits and by its ability to produce perithecia.

Hammarlund (5) described the fungus Erysiphe polyphaga from Veronica speciosa. He found that species to be capable of parasitizing 62 of 100 species, including Cucumis melo and C. sativus, in 11 plant families. He considered the cucurbit and dahlia strains of E. cichoracearum, as well as several species of Oidium from several hosts, to be identical with E. polyphaga. Hammarlund observed abundant perithecia on V. speciosa and a few on some other hosts but none at all on most of the hosts that he used. His work was confirmed by Blumer (2, 3) who added four plant families to the host range of E. polyphaga. Hammarlund's fungus attacked tomato, Lycopersicon esculentum, and several other host species that are not attacked by the cucurbit powdery mildew fungus in southern California. The relationship of his fungus to the latter must await further work.

The fungus reported here is considered to be a conidial clone of an unknown species of the Erysiphaceae since it has not been observed to produce perithecia. The conflicting host-range reports in the literature, the demonstration by Jagger, Whitaker and Porter (6) of two biolog-

ical races in southern California, and the demonstration of strains with different temperature requirements by Yarwood, et al. (18), all suggest that the relationships among cucurbit powdery mildew fungi need clarification. The demonstrations of heterothallism in Erysiphe cichoracearum on sunflower by Yarwood (17) and on lettuce by Schnathorst (14) suggest that the strains on cucurbits may be plus or minus clones of a heterothallic parasite. It seems desirable that studies designed to determine the true identity, or identities, of Oidium stages of Erysiphaceae on cucurbits should be made.

The findings (here and 7) that conidial clones of the cucurbit powdery mildew fungus that attacks cucurbits in southern California can attack such unrelated species as Eremocarpus setigerus and Carica papaya demonstrate that the host range of this fungus extends beyond the Cucurbitaceae and may extend much farther.

The establishment of successful parasitic relations by the cucurbit powdery mildew race 2 fungus on the turkey mullein, a weed indigenous to the foothills near the irrigated desert valleys of the southwestern United States, suggests that this, and perhaps other, weed species may have an important role in the life history and dissemination of the parasite in the important spring cantaloupe-producing areas of the Southwest.

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# PINK ROOT DISEASE OF ONIONS IN MENDOZA, ARGENTINA

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During the last two growing seasons (1959-60; 1960-61), the pink root disease of onions (Allium cepa), caused by Pyrenochaeta terrestris (Hans.) Gorenz, J. C. Walker, & Larson[2], came into prominence in several fields in the province of Mendoza, Argentina.

A great number of mycelial isolates of P. terrestris were obtained by plating infested roots in potato-dextrose agar and Czapek medium, after washing thoroughly in water, and then immersing for 3 to 5 minutes in calcium hypochlorite solution[3]. Pure cultures of the organism were often obtained, but in several cases other fungi appeared from the roots in close association with the pink root organism. The colonies of the latter could be recognized by their relatively slow, compact growth, by the greyish aerial mycelium, by the occurrence of pycnidial primordia and by comparison with cultures kindly sent to us by Dr. R. H. Larson of the University of Wisconsin and Dr. E. C. Tims of Louisiana State University.

Until the present time, no production of sporulating mutants in mycelial lines was observed. The disease has been artificially reproduced in the greenhouse following the method described by Gorenz, et al.[4] and the causal fungus re-isolated.

This appears to be the first time that the pink root disease of onion has been reported in Argentina.

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## OCCURRENCE OF PUCCINIA POLYSORA IN ILLINOIS

A. L. Hooker

Puccinia polysora Underw. was found for the first time in Illinois in 1958. Telia were present on leaves of dent corn collected in Fayette County in south-central Illinois in September. The infection was light and mixed with that of Puccinia sorghi Schw. Specimens have been deposited in the herbarium of the Illinois Natural History Survey, Urbana, Illinois and in the Arthur Herbarium, Lafayette, Indiana.

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## RESULTS OF 1960 FUNGICIDE AND NEMATOCIDE TESTS

The "Results of 1960 Fungicide and Nematocide Tests" is now available. This report is issued annually by the American Phytopathological Society, Subcommittee on New Fungicide and Nematocide Data. This report serves as a medium for organizing and presenting the summarized results of current fungicide and nematocide testing projects. Much of the information is never otherwise published or made conveniently available. Information on products available for testing, composition of products and their sources are given.

Copies of this report are available at $1.00 per copy when accompanied by a remittance, $1.25 when invoiced and billed. Address orders to A. B. Groves, Winchester Fruit Research Laboratory, 2500 Valley Avenue, Winchester, Virginia. Make remittances payable to the American Phytopathological Society.

## ANNOUNCEMENT

In response to many recent inquiries, we should like to tell the readers of the Plant Disease Reporter that with publication of Supplement 262 the Supplement Series was discontinued. -- P. R. Miller

## CORRECTION

REPORTER, January issue (Volume 45, Number 1). On page 66, first paragraph, change line 2 to read: "given 100% control of R. similis in. . . . etc."

The Reporter regrets the omission of the word control.